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Association between strained capacity and mortality among patients admitted to intensive care: A path-analysis modeling strategy☆

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ABSTRACT

Purpose: To evaluate the associations between strained ICU capacity and patient outcomes.
Methods: Multi-center population-based cohort study of nine integrated ICUs in Alberta, Canada. Path-analysis modeling was adopted to investigate direct and indirect associations between strain (available beds ≤1; occupancy ≥95%) and outcomes. Mixed-effects multivariate regression was used to measure the association between strain and acuity (APACHE II score), and both acuity and strain measures on ICU mortality and length of stay.
Results: 12,265 admissions comprise the study cohort. Available beds ≤1 and occupancy ≥95% occurred for 22.3% and 17.0% of admissions. Lower bed availability was associated with higher APACHE II score ($p < 0.0001$). The direct effect of ≤1 available beds at ICU admission on ICU mortality was 11.6% (OR 1.116; 95% CI, 0.995–1.252). Integrating direct and indirect effects resulted in a 16.5% increased risk of ICU mortality (OR 1.165; 95% CI, 1.036–1.310), which exceeded the direct effect by 4.9%. Findings were similar with strain defined as occupancy ≥95%. Strain was associated with shorter ICU stay, primarily mediated by greater acuity.
Conclusions: Strained capacity was associated with increased ICU mortality, partly mediated through greater illness acuity. Future work should consider both the direct and indirect relationships of strain on outcomes.

1. Introduction

Strain on intensive care unit (ICU) capacity is conceptually defined as a dynamic discrepancy between the availability of ICU resources (i.e., beds, ventilators, clinicians) and demand to admit and provide high-quality care for patients with critical illness [1,2]. Strained capacity is perceived among clinicians to be encountered more frequently due to growing demand for and relatively fixed supply of critical care services [1].

Strained ICU capacity may contribute to suboptimal quality of care [1,3], may modify clinician behaviors and care processes [4,5], and may increase susceptibility for adverse events [6], premature ICU discharges [7], unplanned readmissions [8] and mortality [9]. Recent observations suggest sustained strain may negatively impact clinician well-being [10,11].

Numerous factors contribute to strain including bed availability [4,12], bed turnover rate, patient acuity [9], bedside workload [13,14] and bed-block (i.e., discharge limitation) [15]. Selected measures of strain have been characterized (e.g., census, acuity, new admissions [8,9]); however, studies have not specifically evaluated the impact of strained ICU capacity in a large integrated health jurisdiction where the care of critically ill patients is coordinated across ICUs [16,17]. Moreover, their generalizability to health regions with considerably lower numbers of ICU beds is uncertain [18-20]. Finally, most studies have examined the direct relationships (typically logistical regression) between

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measures of strain and outcomes [8], but have seldom explored potential indirect effects of strain (e.g., mediation through illness acuity) on outcomes. Prior work has suggested a complex relationship may be indirectly mediated between strain and ICU care processes [5].

To begin to characterize this complex relationship between strain measures and patient outcomes, we performed a population-based cohort study using path-analysis integrating both the direct and indirect effects strain may exert on outcomes. The work was conducted in a large geographically-defined healthcare system serving a population of approximately 4 million residents in Alberta, Canada (estimated 7.9 ICU beds/100,000 population [20]), where ICUs regularly operate at near or full capacity with often marginal reserves to manage planned admissions, day-to-day variability, and unanticipated surges in demand.

We hypothesized that increased strain on ICU capacity would be associated with greater risk of ICU and hospital mortality. We further hypothesized that strained capacity may exert both direct and indirect influences on risk of death. As such, we hypothesized that direct effects will be shown with selected strain measures (i.e., occupancy, bed availability), while indirect effects will be mediated through variation in illness acuity.

2. Materials and methods

2.1. Study design, setting, and population

This was a population-based cohort study of 9 adult ICUs in Alberta, Canada between June 19, 2012 and December 14, 2014. All ICUs were mixed medical/surgical units in the two major cities in Alberta: Calgary (4 units) and Edmonton (5 units). Of these, 2 are classified as academic/tertiary, 2 as tertiary, and 5 as metropolitan/community ICUs (Supplementary Tables 1 and 2). All included ICUs were staffed by board certified intensivists, had in-house coverage by clinical associates or resident trainees, and had availability of after-hour intensivist coverage. All consecutive adults (age ≥ 15 years) admitted to any of the 9 ICUs were eligible for inclusion.

2.2. Data sources

We analyzed data from a provincial clinical information system (*eCritical Alberta*) coupled with a data warehouse and clinical analytics system (*TRACER Alberta*) (http://www.albertahealthservices.ca/assets/about/scn/ahs-scn-sb-cc-ecritical.pdf). During the study period, six of the study ICUs were implemented on *eCritical*. As such, these ICUs were did not contribute data for the full study period (Supplementary Tables 1 and 2). For the purpose of ensuring the reliability of data from newly initiated ICUs, we omitted the first month contributed to *eCritical*.

eCritical is composed of a bedside system (MetaVision™, iMDsoft, Germany), which provides for full electronic inter-disciplinary clinical documentation and collation of demographic, diagnostic/case-mix (i.e., comorbidity, diagnostic classification, surgical status, Acute Physiology and Chronic Health Evaluation [APACHE] II and III score), laboratory and device data (i.e., monitor, ventilator, vasoactives, renal replacement therapy). *TRACER* provides a comprehensive, multimodal and integrated data repository of patient-specific ICU data enabling creation of reports and specific data extracts for administrative, quality, and research purposes. *eCritical* and TRACER are governed by rigorous methods of data quality assurance and audit. The *eCritical* platform functions within Alberta Health Services (AHS), governed by a provincial multi-disciplinary executive leadership group. *eCritical/TRACER* has previously been used to support health services research [4,21]. Charlson comorbidity index score was ascertained by linkage of *eCritical* data with AHS Discharge Abstract Data (DAD) housed in the AHS Data Repository for Reporting [22].

2.3. Exposures and outcomes

Primary exposure was ICU strain, defined as instantaneous bed availability (≤1, ≤2 or ≤3 beds available at the time of patient admission) and as instantaneous bed occupancy (≥90%, ≥95% proportion of occupied beds at the time of patient admission). While additional measures of strain have been proposed [8,9], we have focused this study specifically on "bed-side" factors (i.e., occupancy, bed availability) in recognition that these are more likely to have immediate influence on clinician decision-making about ICU admission, rather than "resource-side" factors present in the ICU (i.e., new admissions, average acuity, bedside workload). APACHE II score was automatically calculated from data acquired by eCritical in the first 24 h following ICU admission. Primary outcome was all-cause ICU mortality. Secondary outcomes were hospital mortality and ICU length of stay (LOS).

2.4. Statistical analysis

Data were initially explored descriptively. Normally or near normally distributed data were reported as means with standard deviations (SD) and compared by Student's *t*-test. Non-normally distributed continuous data were reported as medians with inter-quartile ranges (IQR) and were by Mann–Whitney *U* test. Categorical variables were compared using the Chi-squared test.

2.4.1. Path-analysis model

We developed a path-analysis model to estimate the magnitude and significance of hypothesized causal associations (direct, indirect and the total combined effect of both) between measures of strained capacity and outcomes (Fig. 1). We first measured the association of measures of strained capacity on patient illness severity at ICU admission (APACHE II score) using a mixed-effects linear regression model, adjusted for covariates that could confound the association between illness severity and strained capacity: age, sex, pre-existing comorbid disease, site (city, hospital type [academic, tertiary and community]), primary diagnostic category, admission category (i.e., medical, neurological, surgical, trauma), surgical status (i.e., elective, emergency), and after-hours admission (i.e., between 22:00 to 7:00). The intent of this analysis was to understand how much the relative contribution of the admission APACHE II score was attributed to strained ICU capacity, assuming random intercepts across the 9 ICUs. We then measured the direct effects of strain measures on ICU mortality, hospital mortality and ICU LOS using a mixed-effects logistic/log-linear regression model, similarly adjusted for the covariates listed above. To integrate the total effect of both the

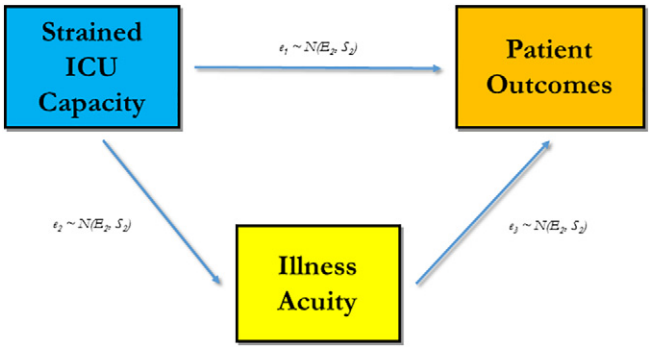

Fig. 1. Path diagram showing the hypothesized causal direct and indirect associations between strain, illness acuity and outcomes. **Footnote**: After modeling, we extracted three pairs of parameters (E_i, S_i), $i = 1,2,3$ (estimate, standard error), which were used to describe the direct and indirect associations of ICU capacity strain to patient outcomes (i.e., ICU mortality). In detail, we used normal distribution $N(E_1,S_1)$ to describe the direct association, and $N(E_2,S_2)$ and $N(E_3,S_3)$ to describe the indirect associations. We did a simulation with 1 million replications to calculate indirect and total (integrated direct and indirect) effects using parameters and the following algorithm: **Algorithm 1**. Estimate indirect and total (integrated) effect of ICU capacity strain.

Table 1
Summary of characteristics of patients and occupancy variables at ICU admission, stratified by ICU vital status.

Characteristic	Total (n = 12,265)	Survived ICU (n = 10,463; 85.3%)	Died in ICU (n = 1802; 14.7%)	P Value
Age, median (IQR)	59 (46–70)	58 (44–69)	64 (52–75)	<0.0001
Age category, n (%)				<0.0001
<65 years	7680 (62.64)	6774 (64.77)	906 (50.28)	
65–74 years	2458 (20.05)	2019 (19.31)	439 (24.36)	
75–84 years	1696 (13.83)	1369 (13.69)	327 (18.15)	
≥85 years	426 (3.47)	296 (2.83)	128 (7.21)	
Sex, n (%)				0.4769
Female	5112(41.69)	4347 (41.56)	765 (42.45)	
Male	7150 (58.31)	6113 (58.44)	1037 (57.55)	
Location, n (%)				<0.0001
Calgary	6732 (54.89)	5641 (53.91)	1091 (60.54)	
Edmonton	5533 (45.11)	4822 (46.09)	711 (39.46)	
Primary diagnostic system, n (%)				<0.0001
Cardiovascular	2103 (17.26)	1496 (14.40)	607 (33.82)	
Gastrointestinal	2044 (16.78)	1700 (16.36)	344 (19.16)	
Genitourinary	449 (3.69)	423 (4.07)	26 (1.45)	
Hematology	65 (0.53)	52 (0.50)	13 (0.72)	
Metabolic/endocrine	246 (2.02)	238 (2.29)	8 (0.45)	
Musculoskeletal/skin	485 (3.98)	443 (4.26)	42 (2.34)	
Neurologic	1809 (14.85)	1648 (15.86)	161 (8.97)	
Respiratory	3643 (29.90)	3176 (30.57)	467 (26.02)	
Transplant	129 (1.06)	126 (1.21)	3 (0.17)	
Trauma	1211 (9.94)	1087 (10.46)	124 (6.91)	
Surgery, n (%)				<0.0001
Elective	1209 (9.86)	1183 (11.31)	26 (1.44)	
Emergency	2054 (16.75)	1827 (17.46)	227 (12.60)	
Non-operative	9002 (73.40)	7453 (71.23)	1549 (85.96)	
Admission category, n (%)				<0.0001
Medical	7241 (59.04)	5985 (57.20)	1256 (69.70)	
Neurological	851 (6.94)	678 (6.48)	173 (9.60)	
Surgical	3101 (25.28)	2855 (27.29)	246 (13.65)	
Trauma (without TBI)	647 (5.28)	607 (5.80)	40 (2.22)	
Trauma (with TBI)	425 (3.47)	338 (3.23)	87 (4.83)	
Site-specific ICU, n (%)				<0.0001
Academic 1	3083 (25.14)	2552 (24.39)	531 (29.47)	
Academic 2	333 (2.72)	292 (2.79)	41 (2.28)	
Tertiary 1	303 (2.47)	253 (2.42)	50 (2.77)	
Tertiary 2	1728 (14.09)	1436 (13.71)	292 (16.20)	
Community 1	1936 (15.78)	1695 (16.20)	241 (13.37)	
Community 2	1258 (10.26)	1072 (10.25)	186 (10.32)	
Community 3	268 (2.19)	273 (2.27)	31 (1.72)	
Community 4	663 (5.41)	581 (5.55)	82 (4.55)	
Community 5	2693 (21.96)	2345 (22.41)	348 (19.31)	
ICU size, median (IQR)	24 (18–28)	24 (10–28)	25 (18–28)	0.0001
Hospital type				0.2113
Academic	5776 (47.09)	4897 (46.80)	878 (48.78)	
Tertiary	3664 (29.87)	3131 (29.92)	533 (21.64)	
Community	2825 (23.03)	2435 (23.27)	390 (13.81)	
Comorbidity (yes), n (%)	10,485 (85.49)	8734 (83.48)	1751 (97.17)	<0.0001
Number of comorbidities, n (%)				<0.0001
0	1780 (14.51)	1729 (16.52)	51 (2.83)	
1	3466 (28.26)	3177 (30.36)	289 (16.04)	
2	2729 (22.25)	2315 (22.13)	414 (22.97)	
≥3	4290 (34.98)	3242 (30.99)	1048 (58.16)	
Comorbidity disease, n (%)				
Chronic dialysis	423 (3.45)	350 (3.35)	73 (4.05)	0.1293
Hepatic	1264 (10.31)	882 (8.43)	382 (21.20)	<0.0001
Neurologic	5298 (43.20)	4213 (40.27)	1085 (60.21)	<0.0001
AIDS	65 (0.53)	51 (0.49)	14 (0.78)	0.1180
Chronic heart failure	762 (6.21)	582 (5.56)	180 (9.99)	<0.0001
Respiratory	1415 (11.54)	1174 (11.22)	241 (13.37)	0.0082
Metastatic/leukemia/lymphoma	791 (6.45)	621 (5.94)	170 (9.43)	<0.0001
Immune suppression	1095 (8.93)	877 (8.38)	218 (12.10)	<0.0001
Diabetes	2280 (18.59)	1891 (18.07)	389 (21.59)	0.0004
Cirrhosis	785 (6.40)	584 (5.58)	201 (11.15)	<0.0001
Cardiovascular	5752(46.90)	4478 (42.80)	1274 (77.70)	<0.0001
Digestive	2210 (18.02)	1764 (16.86)	446 (24.75)	<0.0001
Acute renal	2869 (23.39)	2116 (20.22)	753 (41.79)	<0.0001
Admission APACHE II score, mean (SD)	20.23 (8.41)	18.90 (7.67)	28.65 (8.00)	<0.0001
After-hours admissions, n (%)	4251 (34.66)	3576 (34.18)	675 (37.46)	0.0069
Number of available beds, mean (SD)	3.09 (2.26)	3.10 (2.26)	3.03 (2.28)	0.2054
Bed availability, n (%)				
Available beds ≤ 1	2680 (22.32)	2222 (21.76)	458 (25.54)	0.0004

(continued on next page)

Table 1 (*continued*)

Characteristic	Total (n = 12,265)	Survived ICU (n = 10,463; 85.3%)	Died in ICU (n = 1802; 14.7%)	*P* Value
Available beds ≤ 2	5267 (43.87)	4456 (43.63)	811 (45.23)	0.2089
Available beds ≤ 3	7653 (63.75)	6520 (63.85)	1133 (63.19)	0.5939
Bed occupancy rate, median (IQR)	85.71 (79.92–92.31)	85.71 (76.00–92.31)	86.67 (78.57–92.86)	0.0008
Bed occupancy, n (%)				
Occupancy ≥ 90%	4177 (34.79)	3501 (34.28)	676 (37.70)	0.0051
Occupancy ≥ 95%	2041 (17.00)	1691 (16.56)	350 (19.52)	0.0021

Abbreviations: IQR = intraquatile range; SD = standard deviation; ICU = intensive care unit; TBI = traumatic brain injury.

direct and indirectly mediated effects to estimate the overall strength of the hypothesized causal relationship of strained ICU capacity on outcomes, we conducted simulation experiments with 1 million replications performed using R (R Core Team [2015], Austria) (Supplementary Table 3). Using the same analytic method, we performed sensitivity analyses by varying the strain measure at the time of patient ICU admission, defined as available beds ≤2 and ≤3, and bed occupancy ≥90% and ≥95%. We also performed sensitivity analysis on the direct, indirect and total effects of strained capacity on outcomes after adjusting for Charlson comorbidity index score derived from administrative data [22]. All analyses were conducted in SAS (Release 9.3; SAS Institute, NC).

3. Results

There were 12,265 ICU admissions to the 9 ICUs during the study period. Of these, the 2 largest academic ICUs comprised almost half (47.1% of total admissions) (Table 1). The median (IQR) patient age was 59 years (46–70), 58.3% were male, mean (SD) admission APACHE II score was 20.2 (8.4), and 26.7% of admissions were surgery-related (Table 1). ICU mortality was 14.7% (n = 1802).

3.1. Strained ICU capacity measured by bed availability and occupancy

At the time of patient ICU admission, there were ≤1 bed available in 22.3% of cases, ≤2 beds in 43.9%, and ≤3 beds in 63.8%. Similarly, a relative bed occupancy of ≥90% existed in 34.8% of cases, and a bed occupancy of ≥95% was present in 17.0% of cases (Table 1). Site-stratified occupancy and bed availability data showed significant variability (Supplementary Tables 4 and 5).

3.2. Path-analysis modeling for ICU mortality

3.2.1. Factors associated with admission APACHE II score

Available beds ≤1 was associated with a significantly higher admission APACHE II score, suggesting strained capacity was associated with greater acuity at ICU admission (Supplementary Table 6).

3.2.2. Factors directly associated with ICU mortality

In mixed-effects logistic regression, several factors were significantly associated with ICU mortality, including admission APACHE II score (OR 1.091; 95% CI, 1.088–1.098) (Table 2).

3.2.3. Total effect integrating both direct and indirect effects of strain

The direct, indirect, and total effect of strained capacity, as measured by bed availability and occupancy on ICU mortality is shown in Table 3. No strain measure showed a significant association with ICU mortality in direct analysis. However, all measures showed statistically significant effects in indirect analysis, suggesting an increase in ICU mortality mediated by greater patient illness acuity. The total effect, integrating both the direct and indirect effects, showed available bed ≤1 and occupancy ≥95% at ICU admission were both significantly associated with ICU mortality. For example, available beds ≤1 had a direct effect of OR 1.116 (95% CI, 0.995–1.252), an indirect effect mediated through illness acuity of OR 1.044 (95% CI, 1.018–1.070) and total effect of OR 1.165 (95% CI, 1.036–1.310) for ICU mortality. The total effect was associated with a 16.5% incremental risk of ICU mortality, which exceeded the estimated direct effect by 4.9%.

3.2.4. Sensitivity analyses

For strained ICU measures of greater available beds (≤2 and ≤3) and relative occupancy (≥90% and ≥95%), we noted no direct effect on ICU mortality; however, there were significant indirect effects across each measure. The total effect of bed availability and occupancy measures showed a gradient association with ICU mortality (Table 3).

Table 2
Logistic regression results for the association between clinical characteristics, admission APACHE II score and ICU mortality.

Variable	OR (95% CI)	*p*-Value
Intercept	–	<0.0001
Available beds ≤ 1	1.116 (0.995, 1.252)	0.0585
Age		
Age < 65 years		
Age 65–74 years	1.096 (0.969, 1.241)	0.1460
Age 74–84 years	1.171 (1.018, 1.347)	0.0267
Age ≥ 85 years	1.947 (1.588, 2.388)	<0.0001
Hospital type		
Academic	–	
Community	5.089 (2.174, 11.912)	0.0100
Tertiary	1.596 (0.583, 4.367)	0.3999
Primary diagnostic organ system		
Cardiovascular	–	
Gastrointestinal	0.861 (0.722, 1.026)	0.0950
Genitourinary	0.394 (0.261, 0.596)	<0.0001
Hematology	1.342 (0.730, 2.469)	0.3435
Metabolic/endocrine	0.239 (0.118, 0.481)	<0.0001
Musculoskeletal/skin	0.730 (0.519, 1.027)	0.0708
Neurologic	0.483 (0.386, 0.605)	<0.0001
Respiratory	0.919 (0.801, 1.055)	0.2293
Transplant	0.359 (0.112, 1.146)	0.0838
Trauma	0.532 (0.384, 0.736)	0.0001
Surgery		
Non-operative	–	
Elective	0.363 (0.231, 0.569)	<0.0001
Emergency	0.882 (0.700, 1.110)	0.2855
Admission ICU category		
Medical	–	
Neurological	2.569 (2.066, 3.196)	<0.0001
Surgical	0.911 (0.720, 1.153)	0.4393
Trauma (without TBI)	1.099 (0.721, 1.676)	0.6598
Trauma (with TBI)	2.273 (1.625, 3.178)	<0.0001
Comorbidity		
Chronic dialysis	0.752 (0.585, 0.967)	0.0265
Hepatic	1.485 (1.297, 1.700)	<0.0001
Metastatic/leukemia/lymphoma	1.313 (1.093, 1.577)	0.0036
Immune suppression	0.877 (0.745, 1.031)	0.1121
Cardiovascular	1.267 (1.119, 1.435)	0.0002
Digestive	1.113 (0.964, 1.284)	0.1429
Admission APACHE II score	1.091 (1.085, 1.098)	<0.0001

Abbreviations: IQR = intraquatile range; SD = standard deviation; ICU = intensive care unit; TBI = traumatic brain injury.

Table 3
Total effect of direct and indirect effects of strained ICU capacity on ICU mortality, hospital mortality and ICU length of stay.

Outcome	Direct effect		Indirect effect		Total (integrated effect)	
	OR (95% CI)	*p*-Value	OR (95% CI)	*p*-Value	OR (95% CI)	*p*-Value
ICU morality						
Available beds ≤ 1	1.116 (0.995, 1.252)	0.0601	1.044 (1.018, 1.070)	0.0007	1.165 (1.036, 1.310)	0.0108
Available beds ≤ 2	1.025 (0.928, 1.133)	0.6251	1.037 (1.016, 1.059)	0.0005	1.063 (0.960, 1.178)	0.2385
Available beds ≤ 3	0.948 (0.856, 1.051)	0.3092	1.029 (1.008, 1.052)	0.0082	0.976 (0.879, 1.084)	0.6511
Occupancy ≥ 90%	1.042 (0.936, 1.160)	0.4553	1.047 (1.024, 1.072)	0.0001	1.091 (0.978, 1.218)	0.1207
Occupancy ≥ 95%	1.095 (0.962, 1.247)	0.1692	1.047 (1.017, 1.077)	0.0015	1.146 (1.004, 1.309)	0.0434
Hospital mortality						
Available beds ≤ 1	1.05 (0.955, 1.155)	0.3137	1.033 (1.014, 1.053)	0.0006	1.085 (0.985, 1.195)	0.0992
Available beds ≤ 2	1.041 (0.959, 1.13)	0.3379	1.028 (1.012, 1.045)	0.0006	1.070 (0.985, 1.163)	0.1104
Available beds ≤ 3	0.961 (0.883, 1.045)	0.3530	1.022 (1.006, 1.039)	0.0083	0.982 (0.901, 1.07)	0.6816
Occupancy ≥ 90%	1.017 (0.931, 1.11)	0.7076	1.036 (1.018, 1.054)	0.0001	1.053 (0.963, 1.152)	0.2573
Occupancy ≥95%	1.033 (0.928, 1.15)	0.5561	1.035 (1.013, 1.058)	0.0016	1.069 (0.958, 1.193)	0.2300
ICU length of stay						
Available beds ≤ 1	0.962 (0.922, 1.005)	0.0813	1.010 (1.004, 1.016)	0.0007	0.972 (0.930, 1.015)	0.1974
Available beds ≤ 2	0.953 (0.919, 0.988)	0.0085	1.008 (1.004, 1.013)	0.0005	0.961 (0.926, 0.996)	0.0304
Available beds ≤ 3	0.953 (0.918, 0.989)	0.0118	1.007 (1.002, 1.012)	0.0083	0.959 (0.924, 0.996)	0.0313
Occupancy ≥ 90%	0.951 (0.915, 0.990)	0.0136	1.010 (1.005, 1.016)	0.0001	0.961 (0.924, 1.001)	0.0536
Occupancy ≥ 95%	0.943 (0.898, 0.991)	0.0203	1.010 (1.004, 1.017)	0.0015	0.953 (0.907, 1.002)	0.0592

3.2.5. Sensitivity analysis for Charlson comorbidity index

After data linkage for calculation of Charlson comorbidity index, only 11,361 patients (92.6%) were included in multivariable modeling. The median (IQR) Charlson comorbidity index was 1 (0–2) and showed significant association with ICU mortality ($p < 0.0001$). In multivariable analysis, Charlson comorbidity index was significantly associated with admission APACHE II score ($p < 0.0001$). The total effect, integrating the direct and indirect effects following adjustment for Charlson comorbidity index, showed strained ICU capacity, defined by ≤1 bed available, remained significantly associated with increased ICU mortality (OR 1.164; 95% CI, 1.035–1.310, $p = 0.0112$).

3.3. Path-analysis modeling for secondary outcomes

3.3.1. Hospital mortality

Bed availability and occupancy measures of strained ICU capacity showed no direct or total effect with hospital mortality, whereas indirect effects were all statistically significant (Table 3).

3.3.2. ICU length of stay

Bed availability and occupancy measures of strained capacity showed mixed effects in direct and indirect analyses (Table 3). The total effect of strained capacity measures implied higher illness acuity was associated with a shorter ICU stay. For bed availability ≤2 and ≤3, the total effect implied a reduction in ICU stay (3.9% and 4.1% for each, $p = 0.0304$, $p = 0.0313$).

4. Discussion

4.1. Key findings

In this study, we used an innovative two-stage approach to evaluate both the direct and indirect associations of common measures of strained ICU capacity and ICU mortality in a publicly-funded integrated health region. We found approximately 1 in 5 admissions to ICU occurred under circumstances of strained capacity when defined by low bed availability and/or high occupancy at the time of admission. We also showed the prevalence of strain varied across ICUs, generally being higher in academic/tertiary ICUs. Strained capacity was associated with greater illness severity among patients admitted. ICU mortality was found to be greater for admissions occurring during periods of strained capacity. The total effect of strained capacity on ICU mortality, integrating both the direct and indirect effects, exceeded the direct effect alone and showed a gradient response with reduced bed availability at the time of admission. Finally, in integrated analysis considering indirect effects mediated though illness acuity, strained capacity was associated with reduced length of ICU stay.

4.2. Interpretation with prior work

Demand for critical care services is projected to increase. A Canadian population-based study found admissions to ICU had contracted; however, the average length of ICU stay had increased, suggesting sustained growth in ICU bed utilization over time [17].

Lack of bed availability, as a measure of strain, has not only forecasted greater risk for ICU mortality, but also reduced likelihood of ICU admission among patients having an acute deterioration on the ward [4]. Notably, denial of ICU admission has not consistently translated into increased hospital mortality, implying there is likely significant practice variation in ICU resource utilization and provider uncertainty in selecting patients perceived to most likely to derive benefit from ICU support [4,23-25]. Comparable to our findings, reduced bed availability has been associated with greater illness severity, implying strain influences decision-making around triage suitability or rather, provokes queuing contingent on subsequent clinical deterioration [9,12]. Similarly, withdrawal of life-sustaining therapy may be more likely during periods of strain, which may account for both higher ICU mortality and indirectly, shorter ICU stay [5].

Consistent with prior data, we also found shorter ICU stay during periods of strain [8]. Reduced ICU stay was likely partly mediated through both greater ICU mortality and earlier ICU discharge, and transition to the ward among survivors. Moreover, in select small community ICUs, we observed a high proportion of patients were discharged directly home from the ICU. These observations provide compelling circumstantial evidence that health systems contribute to avoidable ICU-bed days and/or can surge expansion of ward bed capacity in response to ICU strain. Strain, as defined by bed availability in our study, is also clearly modified by bed-block and patient flow failure from the ICU. However, strain has negative implications for patient care on the ward and in the emergency department (ED). Reduced bed availability has shown incremental risk of ward cardiac arrest and ICU readmission [26]. Boarding in the ED has been associated with adverse outcomes [27,28] and delayed admission due to strained capacity associated with increased mortality [12].

These observations highlight the patient-level and system-level tension between ICU capacity and operational efficiency [29,30]. Moreover, facets of strain may partially constitute waste and overuse of available ICU beds [23,24]. The availability of excess or “empty” ICU beds begets their use, particularly in jurisdictions where hospital remuneration

drives utilization [30]. At the same time, clinicians have identified inappropriateness of ICU utilization as a modifiable contributor to strain [1].

4.3. Implications for policy, practice and research

Our study implies that there are measurable consequences to strained ICU capacity. While these effects may be perceived as relatively small, these have significant implications for ICU capacity and organizational planning. In recent years, considerable investment in ICU resources has been directed beyond traditional ICU settings to "rescue" patients deteriorating on hospital wards. However, examples of widespread implementation of rapid response systems have not consistently been proven to improve outcomes or translate into improved health system efficiency [31]. We also contend that during periods of strain, health systems need to identify the mechanisms most contributing to strain. Bed availability is undoubtedly an important indicator of strain, particularly for patients in need of ICU care; however, occupancy metrics are also significantly modified by preventable flow failure (i.e., avoidable bed-days and bed-block) [15].

Health systems must develop strategies to manage strain, such as scalable ICU capacity surge expansion of ICU bed capacity and/or ward bed capacity and safely facilitating transition of patients to lower intensity settings. This is essential to enable admission of the "next" critically ill patient in need of advanced life support, while avoiding unnecessary afterhours or premature discharges among those with marginal reserve [7,32]. Similarly, by extension, there should be recognition that strained capacity could also mediate small but not insignificant indirect effects on the risk of adverse outcomes or differential care processes for patients (and their families). In our study, strained capacity was associated with greater ICU mortality, but not with hospital mortality, while also associated with shorter length of ICU stay. Prior data imply increased ICU mortality in response to strain could result from greater rates of withholding or withdrawal of life support among those at greater baseline risk for death, indirectly mediating shorter ICU stay [5]. Likewise, during strain, patients with lower illness acuity may be discharged earlier from ICU with no incremental risk for hospital mortality [8].

Selected jurisdictions have considerable ICU surge capacity due to an abundant ICU bed base [18], variable ICU bed utilization, and lower proportion of mechanically ventilated patients [33]. For example, among the 97 ICUs in the United States-based Project IMPACT database, >50% of ICUs had ≥4 beds available >50% of the time [34]. Accordingly, the complex interplay between strained capacity and outcomes derived from countries with high vs. low numbers of ICU beds and alternative health system funding models may not be comparable.

4.4. Strengths and limitations

Our study has several strengths. It was multi-center integrating comprehensive population-based data from heterogeneous ICUs across an integrated health region. These findings are likely externally valid to similarly organized health jurisdictions; however, may have limited generalizability to those with differing funding models. Our study extends prior work showing important interactions between strained capacity, operational efficiencies and patient outcomes [9,26]. Our study also has limitations. First, our study was retrospective and observational, and may be susceptible to omission of confounders and other sources of bias. Second, following the development of a conceptual model of inputs to strained ICU capacity, we utilized a simplified path-analysis to begin to unravel the complex associations between measures of strain and outcome. In this analysis, we have largely focused on bed availability, occupancy and illness severity, and their association with outcome. As such, we did not concomitantly evaluate additional potential measures of strain that may show association with outcomes, such as clustering of ICU admissions, bedside workload or avoidable-days [6,13]. Third, we did not have operational data on bedside staffing patterns to correlate with strain measures; however, we believe this would be important to integrate into future work. Finally, we did not have data on inter-hospital transfers associated with strain (i.e., transfer from ED to ICUs at separate facilities); and while this may be a strong indicator of strain, this is a relatively uncommon event.

5. Conclusion

In a population-based cohort study of patients admitted to mixed medical/surgical ICUs in a large integrated health region using a two-stage path-analysis strategy, we found small but significant associations between strained ICU capacity, defined by bed availability and occupancy, and ICU mortality and ICU stay. The relationship between strained capacity and outcomes was partly, though not completely, explained by increased illness severity among those admitted during periods of strain. These observations suggest that future assessment of strained capacity, including interventions aimed at averting strain, should consider both the direct and indirect associations strain may have on outcomes.

Declarations

Ethics approval and consent to participate

This study was approved by the research ethics board at the University of Alberta, Edmonton, Canada (File # Pro00046184). The need for written informed consent was waived.

Consent for publication

Not applicable.

Availability of data and material

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

No authors have any conflicts of interest to declare.

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Authors' contributions

SMB conceived the study, obtained funding for the study, obtained data, analyzed the data, interpreted data, drafted the manuscript and provided critical revision of the manuscript. XW analyzed the data, interpreted data and provided critical revision of the manuscript. DAZ interpreted data and provided critical revision of the manuscript. DZ obtained data, interpreted data and provided critical revision of the manuscript. PD interpreted data and provided critical revision of the manuscript. AG interpreted data and provided critical revision of the manuscript. DCS interpreted data and provided critical revision of the manuscript. LB interpreted data and provided critical revision of the manuscript. PF interpreted data and provided critical revision of the manuscript. GC obtained the data, interpreted data and provided critical revision of the manuscript. DO interpreted data and provided critical revision of the manuscript. HTS conceived the study, obtained funding for the study, interpreted data, and provided critical revision of the manuscript.

Conflict of interest

Authors have no conflict of interest to declare.

Initialize: Assign (E_i, S_i), $i = 1, 2, 3$.

Iterate: Repeat following steps 1–2 one million times:

Step 1 created three random numbers from the three normal distributions, i.e. $e_i \sim N(E_i, S_i)$, $i = 1, 2, 3$.

Step 2 calculate direct and integrated effects

$$Indirect\ Effect = e_2{}^{*}e_3,$$

$$Total\ (Integrated) Effect = e_1 + e_2{}^{*}e_3.$$

Summarize: calculate coefficient estimate (mean), SE (standard error), *p*-value (two sided), OR (odds ratio) and its 95% CI (confident interval) of indirect/total effect from the one-million simulated values.

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Appendix A. Supplementary data

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