



Representative case series from New Zealand public hospital admissions in 1998—III: adverse events and death

Robin Briant, John Buchanan, Roy Lay-Yee, Peter Davis

Abstract

Aims. To examine a representative series of adverse events in New Zealand public hospitals where death was the final outcome recorded, with a view to determining the relationship between adverse event and death.

Methods. A review was carried out of the 38 adverse events (AEs) in the New Zealand Quality of Healthcare Study where death was the outcome, and categories of relationship were established. These were identified from the total of 850 AEs determined by two-stage retrospective review of a representative sample of 6579 medical records drawn from 13 NZ public hospitals in 1998. A stricter definition of AEs, comparable with American studies, was then applied to estimate rates of death associated with AEs.

Results. There were 118 deaths at discharge identified in the sample of medical records, giving a rate of 18.0 deaths per 1000 admissions overall. A total of 30 deaths, either at or after discharge, were associated with AEs (4.6 per 1000 admissions); 19 being judged attributable to the AE either “definitely” (10) or probably (9), giving a combined rate of 2.8 AE-attributable deaths per 1000 admissions. The “definite” group had an age, comorbidity, and added-bed-days profile that was close to the average for all deaths associated with an AE. The “probable” group departed from this profile in being younger, exhibiting higher comorbidity, and having twice the added bed-days. Based on population life tables, the average years of life lost was 11.8 years for the definite group and 25.0 years for the probable group. Assessed on the preventability of the associated AE, it was estimated that 2.2 deaths per 1000 admissions were highly preventable. However, once deaths that were not judged to be attributable to the AE were excluded, the rate reduced to 1.3 AE-preventable deaths per 1000.

Conclusions. Because of the nature of the record review procedure used in the New Zealand Quality of Healthcare Study, a finding of death did not necessarily mean that an adverse event and death were causally related. Indeed, it is possible that extrapolations of mortality rates in this and other similar studies over-estimate by about a half the number of deaths caused by healthcare management.

The New Zealand Quality of Healthcare Study (NZQHS)¹ examined 6579 medical records using a two-stage retrospective review applied to a representative sample of hospital admissions for the calendar year 1998. The sample was drawn by systematic list selection (after exclusion of specialist institutions) from 13 public hospitals providing acute care and over 100 beds. The main aim of the Study was to quantify the adverse outcomes of healthcare management in the New Zealand public hospital system.

NZQHS found that 12.9% of public hospital admissions were associated with an adverse event¹ (within the range of the 16.6% and 10.8% found in similar studies from Australia² and the United Kingdom³ respectively). Subsequently, there has been increased awareness of the incidence and outcomes of adverse events. The Institute of Medicine Report⁴ extrapolated from the Harvard Medical Practice Study (HMPS)⁵ and the Utah and Colorado Study (UTCOS),⁶ and estimated the numbers of deaths in the United States from adverse healthcare events. McDonald et al⁷ have challenged the reliability of such extrapolations and Hayward and Hofer,⁸ in a careful examination of deaths attributed to adverse events and sub-optimal care, have argued that the degree of preventability is probably grossly over-estimated.

This paper is the third in a series on clinical aspects of adverse events following those examining medication-⁹ and surgery-related¹⁰ cases. We aim to document in detail the deaths that occurred in patients admitted to New Zealand public hospitals in 1998 for whom an adverse event was identified. Because of the way the study instrument is constructed, the concurrence of an adverse event and death did not necessarily mean that the two were related. The paper seeks, through the careful re-analysis of those adverse events where death was the outcome, to determine the extent of this relationship.

Methods

NZQHS's data collection method has been reported elsewhere.¹ It involved structured implicit review of the medical record, while seeking evidence of harm to patients from healthcare management.

An adverse event (AE) was defined as:

- An unintended injury,
- Resulting in disability, and
- Caused by healthcare management rather than the underlying disease process.

The degree of causation was scored on a scale of 1–6, where 1 is no relation of the injury to healthcare management (and therefore by definition not an AE), 2 is slight association, 6 is definite association, and 3 and 4 are on either side of 50:50.

Preventability of the event, scored on the same scale, was defined as an error in healthcare management due to failure to follow accepted practice at an individual or system level. Patient impact was measured by disability defined as temporary or permanent disability (respectively lasting less or more than 1 year) or death. Attributable or added bed-days refer to those extra days associated with an AE that were spent in the study hospital during one or more admissions.

Nurse screeners and medical reviewers wrote clinical summaries of each case as part of the main study. For the purpose of this re-analysis of deaths, two of the authors (RB and JB) independently studied those summaries and the details of injury, with the aim of discriminating those cases where there was a clear link between the AE and death (from those cases where death was not the result of the identified AE).

Attention was paid to the medical reviewer's scoring of the degree of healthcare causation in the production of injury, but the authors had to make their own determination of the degree of causation of death. A simple categorisation was developed and all cases were placed in one or other category. The authors (RB and JB) then met, and those instances where there was not initial agreement on category, were resolved by discussion and agreement reached.

The categories of relationship established were:

- Death definitely associated with the AE,
- Death probably/possibly associated with the AE, perhaps remote in time,
- Death very unlikely related to AE, or an inevitable death that may have been slightly hastened by the AE, and
- Death unrelated to the AE.

It was not possible to use the same process to determine preventability of a death, as access to only the clinical summaries did not provide the authors with sufficient information. The high degree of preventability of an AE cannot be extended to draw a conclusion on the preventability of the death. However, we have made a sub-analysis of the cases where an AE had both high causation and high preventability scores (4–6), and was detected during the sampled admission; this sub-group matches most closely those patients included in the American studies^{3,6} from which extrapolations to total deaths due to medical error have been made.

Life tables, by age and gender, for the New Zealand population 2000–02¹¹ were consulted to determine the years of life lost by those patients whose deaths were caused by an AE.

Estimated death rates and 95% confidence intervals have been adjusted for the sample design.

Results

In total, 6579 admissions were screened according to set criteria by nurses at the first stage for indications of an AE, with 4119 progressing to second-stage review by physicians using a structured protocol. Of these admissions, 850 were judged to be AEs (12.9% of admissions) that occurred or were detected during the sampled admission, and where there was any evidence of healthcare management causation (scores 2–6). Death was the outcome for 38 of those admissions (4.5% of AEs).¹

In Table 1, the results of the assessment of the causal status of AEs are outlined, together with key patient characteristics. Twenty-four (63%) of the 38 deaths were judged attributable to the AE, either definitely or probably, with 14 considered not related to the reported AE.

Table 1. Causal status of adverse event associated with death, other AEs, all AEs, and all reviewed admissions: characteristics of patients

Causal status	N	Age (years) mean (range)	Age (% <70 years)	Gender (% male)	Maori (%)	Higher deprivation (NZDep96 = 6–10) (%)
A: Definite	11	75.6 (53–91)	36.4% (4/11)	54.5% (6/11)	0% (0/11)	54.5% (6/11)
B: Probable	13	57.9 (31–74)	84.6% (11/13)	46.2% (6/11)	23% (3/13)	76.9% (10/13)
C: Unlikely	8	78.0 (60–91)	75% (6/8)	75% (6/11)	0% (0/8)	50% (4/8)
D: None	6	80.3 (76–87)	0% (0/6)	50% (3/6)	0% (0/6)	83.3% (5/6)
All associated with death	38	70.8 (31–91)	55.3% (21/38)	55.3% (21/38)	7.9% (3/38)	65.8% (25/38)
Other AEs	812	50.7 (0–101)	67.7%	44.3%	16.3%	63.1%
All AEs	850	51.5 (0–101)	66.6%	44.7%	15.9%	63.2%
All reviewed admissions	6579	42.6 (0–104)	76.1%	45.1%	15.4%	62.8%

AE=Adverse event: occurred or was detected during the sampled admission, and any evidence of healthcare management causation (scores 2–6).

Of the deaths definitely or probably associated with the AE, the average age was 75.6 years (Group A – Definite) and 57.9 years (Group B – Probable) respectively; this compared with the average age of 51.5 years for all patients suffering AEs. In the combined Groups A and B, exactly half were males, 62.5% were aged under 70 years,

12.5% were Maori, and two-thirds were from high-deprivation areas of residence. This profile was very similar to that for all AEs.

Key characteristics of the events are outlined in Table 2. For the combined AE-attributable group, the AE causation score was over 50% for nearly all in the group, and the AE preventability score was over 50% for exactly half of them. There were 9.6 mean added bed-days for Group A and 20.6 for Group B, compared with the 9.3 days overall for all AEs.

Comorbidity was slightly below average for Group A and well above for Group B. The patients in Group A (where death was definitely related to the AE) lost an average of 11.8 years of life, while those in Group B (where the AE probably caused death) lost an average of 25.0 years of life.

The preventability of deaths associated with AEs is addressed in Table 3. The data were also analysed using the same criteria as UTCOS⁶ to identify highly preventable AEs; that is, AEs that were detected during the sampled admission and where both the causation and preventability scores were 4 or more.

Applying these criteria, 267 AEs were judged to be highly preventable (4.1% of admissions), of which 15 were associated with death as the outcome (5.6% of highly preventable AEs) (Table 3).

We judged that 9 (60.0%) of the 15 deaths were causally related to the AE, either definitely or probably. Thus, with this calculation, 9 (3.4%) of 267 highly preventable AEs caused death.

Table 4 provides a summary of the distribution of deaths, together with rates of occurrence per 1000 admissions. For all 6579 reviewed admissions in NZQHS, the overall death rate (at discharge) was 1.8%.

The mortality rate was 0.22% (15/6579) where death (either at or after discharge) was the outcome of a highly preventable AE. After re-analysis, the adjusted rate was 0.13% (9/6579), equating to a rate of 1.3 patients per thousand admitted to New Zealand public hospitals.

Table 2. Causal status of adverse event associated with death, other AEs, and all AEs: characteristics of events

Causal status	Number	Charlson Score* mean (range)	Percent AE causation>50%	Percent AE preventability>50%	Added bed-days mean (SD)	Years of life lost (mean)†
A: Definite	11	1.6 (0-7)	100% (11/11)	45.5% (5/11)	9.6 (13.1)	11.8
B: Probable	13	4.0 (0-9)	84.6% (11/13)	53.8% (7/13)	20.6 (28.6)	25.0
C: Unlikely	8	1.5 (0-3)	75% (6/8)	37.5% (3/8)	5.9 (9.0)	9.9
D: None	6	2.2 (0-4)	83.3% (5/6)	50% (3/6)	2.5 (5.6)	8.5
All associated with death	38	2.5 (0-9)	86.8% (33/38)	50% (19/38)	11.5 (19.5)	–
Other AEs	812	1.1 (0-15)	89.3%	36.6%	9.2 (17.0)	–
All AEs	850	1.1 (0-15)	89.2%	37.1%	9.3 (17.1)	–
All reviewed admissions	6,579	0.9 (0-15)	–	–	–	–

AE=Adverse event: occurred or was detected during the sampled admission, and any evidence of healthcare management causation (scores 2-6); *Measure of comorbidity (full range 0-12); Derived from number and seriousness of comorbid conditions identified by study screeners;¹²†Calculated from Life Tables for NZ population 2000–2.¹¹

Table 3. Causal status of AEs associated with death, by preventability of AE

Causal Status	Not preventable	Any preventability*	High preventability†
A: Definite	1	9	5
B: Probable	2	7	4
Death caused by AE	3 (50.0%)	16 (66.7%)	9 (60.0%)
C: Unlikely	1	5	3
D: None	2	3	3
Death not caused by AE	3 (50.0%)	8 (33.3%)	6 (40.0%)
All AEs associated with death (n=30)	6 (100%)	24 (100%)	15 (100%)
Other AEs (n=629)	240	389	252
All AEs (n=659)	246	413	267

AE=Adverse event; was detected during the sampled admission, and high healthcare management causation (scores 4-6); *Preventability scores 2-6; †Preventability scores 4-6.

Table 4. Summary of death rates by causal status and preventability of AE

Variable	Number of deaths*	Death rates (per 1000 admissions) (95% confidence intervals)§
All reviewed admissions (n=6579)	118	18.0 (13.7–22.4)
Admissions associated with AEs	30	4.6 (2.6–6.5)
- Any preventable†	24	3.6 (1.9–5.3)
- Highly preventable‡	15	2.2 (1.1–3.4)
Death associated with AE—not caused by AE	11	1.8 (1.0–2.6)
Death associated with AE—caused by AE	19	2.8 (0.88–4.6)
- Any preventable†	16	2.3 (0.68–3.9)
- Highly preventable‡	9	1.3 (0.17–2.4)

AE=Adverse event; was detected during the sampled admission, and high healthcare management causation (scores 4-6); *All deaths in this table are those associated with AEs (at discharge or some time after discharge), except those for all reviewed admissions (at discharge only); †Preventability scores 2-6; ‡Preventability scores 4-6; §All rates have a common denominator (n=6579); rates and 95% confidence intervals have been adjusted for the sample design.

Group A (definite relationship between AE and death)—There were 11 patients in this group (Table 5). Five of these deaths followed surgery—two cholecystectomies and three operations for carcinoma of the colon (one rendered urgent by colon perforation during diagnostic colonoscopy).

There were two fatal infections: a *Clostridium difficile* colitis case following antibiotic for exacerbation of COPD, and an under-treated staphylococcal bloodstream infection associated with a pacing wire.

Two patients died before their diagnosis was established, one in-hospital patient with high probability of thromboembolism not adequately investigated or treated, and one community patient with high risk for ischaemic heart disease where chest pain was not acted upon and an untreated myocardial infarction progressed to cardiogenic shock.

One patient with many comorbidities and an abdominal aortic aneurysm (AAA) waited 3 months on an urgent tertiary centre vascular waiting list; then when rupture

occurred, waited many hours for surgery at the peripheral hospital and died before the operation began.

One frail resthome resident fell fracturing the neck of femur (falls in rest homes or other health care facility were classed as AEs) and died of acute haematemesis 3 weeks after the fracture and its fixation.

Group B (probable/possible relationship between AE and death)—This group comprised 11 patients (Table 6). Several patients are included in this group rather than Group A because (although there was a close time relationship between a procedure and death) the cause of death was not clarified in the case records. For two of these patients, a very long lag occurred between relevant healthcare procedure and death.

In this group, there were four deaths related to surgery and two deaths related to diagnostic procedures. There was one probable infection in the remaining lung of a patient after pneumonectomy, and one probable myocardial infarction after partial hepatectomy in another patient.

A patient with carcinoma of the colon experienced major postoperative complications, and another patient had persistent subacute bowel obstruction and died 6 months after complicated pelvic surgery. There was one fatal myocardial infarction after bronchoscopy. One patient with end-stage renal failure on dialysis and many comorbidities had a liver biopsy to evaluate abnormal enzymes and died a few days later with a hepatic subcapsular haemorrhage, ascites, and pleural effusions.

There were two cases of non-diagnosis and one of late diagnosis. A patient with typical cardiac pain was discharged early from a hospital emergency department after a single normal electrocardiogram (ECG), returned later with a completed myocardial infarction too late to thrombolysed, and died after 2 months of severe cardiac failure.

One patient died suddenly probably from pulmonary embolism from a brachial venous thrombosis after arm compression in a community drug coma. One had melanoma, mistaken for a benign lesion, so referral for excision was not made urgent, and 5 months elapsed before the diagnosis and excision; the patient presented with cerebral metastases 1 year after surgery and died soon after.

There were two infective complications in complex cases, one a young person with severe asthma requiring repeated ICU admissions and one with aspiration pneumonia. Two patients had very late deaths; one gastric bypass operation for obesity in 1968 was probably responsible for cirrhosis and hepatic failure in 1998, and a woman who had cervical biopsy for carcinoma *in situ* in 1973 had no follow-up smears and died of invasive carcinoma of cervix in 1998.

Table 5. Group A cases (n=11)—clinical characteristics of individual adverse events

Age	Condition/procedure	AE	Cause of death	Time from AE to death
81	Ca colon, right hemicolectomy	Anastomotic leak	Sepsis	3 days
84	Ca colon, right hemicolectomy	Epidural, hypotension, MI	MI	4 days
72	Ca colon, colonoscopy, partial colectomy	Perforation of colon at colonoscopy, urgent surgery	Anastomotic breakdown, sepsis	4 months
81	Laparoscopic cholecystectomy	Haemorrhage	Complications of haemorrhage	2 days
81	Open cholecystectomy	Bile duct trauma	Persistent bile leak, sepsis	4 months
70	Abdominal aortic aneurysm rupture	Delay for elective and acute surgery	Aortic rupture	Elective 3 months Acute 7 hours
83	Heart block, pacing wire	Pacing wire infection, inadequate treatment	Staphylococcal sepsis	Days
71	Exacerbation COPD, antibiotic	<i>Clostridium difficile</i> colitis	Toxicity, dehydration	1 week
65	Venous thromboembolism	Non-diagnosis (hospital)	Pulmonary embolism	3 days
53	Myocardial infarction	Non-diagnosis (GP)	Cardiogenic shock	1 week
91	Fracture neck of femur	Fall in Rest home	Haematemesis post-operation	3 weeks

AE=Adverse event; occurred or was detected during the sampled admission, and any evidence of healthcare management causation (scores 2–6); Ca=Cancer; COPD=Chronic obstructive pulmonary disease; MI=Myocardial infarction.

Table 6. Group B cases (n=13)—clinical characteristics of individual adverse events

Age	Condition/procedure	AE	Cause of death	Time from AE to death
64	Lung Ca, pneumonectomy	Probable infection remaining lung	Infection	3 days
60	Hepatoma, partial hepatectomy	Probable myocardial infarction	Myocardial infarction	1 day
74	Ca Colon, right hemicolectomy	Fluid overload bladder fistula	Left ventricular failure	6 days
67	Bladder and uterine cancer, resection	Persistent subacute small bowel obstruction	Complications of surgery and obstruction	6 months
56	ESRF on dialysis, abnormal liver enzymes and comorbidities, liver biopsy	Subcapsular haemorrhage	Subcapsular haemorrhage and pleural effusions	5 days
67	Possible lung cancer, bronchoscopy	Myocardial infarction	Myocardial infarction	3 days
45	Arm compression following drug overdose, probable brachial vein thrombosis	Non-diagnosis	Probable pulmonary embolism	10 days
65	Myocardial infarction	Non-diagnosis (hospital ED)	Myocardial infarction, heart failure	2 months
31	Melanoma	Late diagnosis	Cerebral metastases	18 months
33	Asthma, prolonged admission ICU	Nosocomial septicaemia	Multiorgan failure	4 days
73	COPD, poor swallow, NG tube	Delayed jejunostomy, aspiration pneumonia, inadequate treatment	Respiratory failure, probable lymphangitis carcinomatosis	Days
51	Obesity, gastric bypass surgery 1968	Steatosis > cirrhosis	Hepatic failure	Many years
67	Cervical Ca <i>in situ</i> , cone biopsy 1973	No follow up	Invasive cervical cancer	25 years

AE=Adverse event: occurred or was detected during the sampled admission, and any evidence of healthcare management causation (scores 2–6); Ca=Cancer; COPD=Chronic obstructive pulmonary disease; ED=Emergency department; ESRF=End-stage renal failure; ICU=Intensive care unit; NG=Nasogastric.

Group C (unlikely relationship between AE and death)—There were eight patients in this group (Table 7). Although these patients suffered an AE, either it was judged that there was no connection between AE and death, or the AE did no more than hasten inevitable and imminent death.

Five of the patients, aged 83–91 years, were described as frail and they suffered a range of terminal events where healthcare causation may have played a small part in death—a fall in a rest home, a fall from a hospital bed, a delay in inserting a gastrostomy leading to aspiration pneumonia, delay in sorting out a complex presentation, and complications of cardiovascular medication. There was one patient with advanced malignancy, one with terminal muscular dystrophy; and one with myelodysplasia and pneumonia for whom appropriate healthcare actions may have hastened imminent death.

Group D (no relationship between AE and death)—There were six patients in this group (Table 8) where the authors could not identify any connection between healthcare management and death and, in some instances, could not confirm that an AE had occurred.

One demented patient with atrial fibrillation had warfarin stopped (in consultation with family) after risk-benefit assessment; she died 6 months later of a cerebral embolism.

One acutely-admitted patient died before he could have upper gastrointestinal endoscopy—but death was due to small bowel infarction, and there would have been no benefit from the endoscopy.

Two patients had falls at home and died within hours; one of an acute subdural haematoma before he could be taken to theatre, and one had an extensive myocardial infarction earlier which probably caused the fall. The remaining two patients died 1 and 5 months after surgery, but there was no relationship between the surgery and the death.

Table 7. Group C cases (n=8): clinical characteristics of individual adverse events

Age	Condition/procedure	AE	Cause of death	Time from AE to death
60*	End-stage cardiomyopathy	Delayed out-patient appointment	Heart failure, digoxin toxicity, VF arrest	Days
84*	Fracture neck of femur, advanced heart and renal failure	Fall in Rest Home	Pre- and post-operative heart failure	8 days
86	Oesophageal stricture and dysmotility	Delay in gastrostomy insertion, intravenous saline overload	Aspiration pneumonia, Left ventricular failure	1 month
86	Severe anaemia, delirium	Increased sedation with Heminurin	Cause not established	1 week
62*	End-stage muscular dystrophy, chest infection, fall at home # NOF	Morphine and high flow oxygen in Ambulance	Respiratory failure	Hours
70*	Terminal metastatic bladder cancer, severe haematuria	Staphylococcal septicaemia, following catheter and iliac artery embolisation	Staphylococcal sepsis, renal failure	4 days
91	Frail, advanced heart failure. Rest home resident	Renal failure on diuretic and ACE inhibitor, digoxin toxicity	Renal failure, digoxin toxicity	Days
85*	Myelodysplasia, pneumonia, delirium	Fall out of hospital bed	Fracture femur, pneumonia, neutropaenia	4 days

AE=adverse event: occurred or was detected during the sampled admission, and any evidence of healthcare management causation (scores 2-6).;* Considered inevitable deaths where AE may have hastened death slightly; NOF=Neck of femur; ACE=Angiotensin-converting enzyme; VF=Ventricular fibrillation

Table 8. Group D cases (n=6)—clinical characteristics of individual adverse events

Age	Condition/procedure	AE	Cause of death	Time from AE to death
81	Peri-prosthetic fracture femur, inoperable. Acute bleeding gastric ulcer, oversewing. Myocardial infarction	Postoperative MI	Complete heart block, asystolic arrest	2 weeks
78*	Haematemesis	Delay in acute gastroscopy	Massive gut necrosis	1 day
77*	Dementia, atrial fibrillation, CVA	Warfarin discontinued (done with discussion because of dementia)	New CVA, presumed embolic	5 months
87	Chronic gangrenous ulcers foot and leg, amputation. Dementia, heart failure	Postoperative pneumonia	Infection, dehydration	4 months
83*	Fractured femur, fall at home. Acute myocardial infarction, bradycardia/sick sinus syndrome	Pacemaker insertion, pacing failed	Asystolic cardiac arrest	3 hours
76*	On warfarin for AF, fall at home, hit head. Hours later rapid loss of consciousness, inoperable subdural haematoma.	Failure of GP to admit at initial consultation	Acute subdural haematoma	1 day

AE=Adverse event; occurred or was detected during the sampled admission, and any evidence of healthcare management causation (scores 2–6); *The authors did not confirm that an AE had occurred; AF=Atrial fibrillation; CVA=Cerebrovascular event; MI=Myocardial infarction.

Discussion

Because of the way the study instrument functions, one cannot necessarily conclude that the death is causally linked to the identified AE. Therefore, we have reassessed carefully all aspects of the information provided and made a determination on the causal relationship in each case.

Key findings—The numbers are very small, and so the information is indicative only. Our re-analysis showed that 14 of 38 deaths were found to have a very tenuous link, or none at all, with healthcare causation. This makes the other reported deaths from similar AE studies at least slightly suspect and probably an overestimate of the fatal impact of AEs.

Based on the American definition of an AE, we concluded that 19/659 AEs (2.9%) led to death. In our studied population of 6579 patient admissions, the death rate due to healthcare management was 0.28% (19/6579), or 2.8 healthcare-related deaths per 1000 patients admitted to New Zealand public hospitals; the overall death rate was 1.8% (118/6579). Furthermore, three-fifths of highly preventable AEs where death was the outcome (9/15) directly caused the deaths of 1.3 per 1000 New Zealanders admitted to hospital.

Strengths and limitations of this study—As far as we are aware, this is the first in-depth examination of deaths from an AE study using retrospective chart review, and it calls into question the estimations of death rates drawn from other similar AE studies. The main utility of studies of AEs and their outcome is to provide a basis for understanding how we can improve the healthcare system and reduce the amount of disability and the number of deaths from medical misadventure.

To this end, the question of the preventability of AEs is crucial, but alas it is also the most difficult aspect of the study. Thus the reviewer must ‘read between the lines’ as well as carefully study the written record of the patient’s hospitalisation to understand the process of care and determine if there were errors or omissions in management, meaning that another standard of care might have prevented the mishap.

In this analysis, the primary sources of information were not available and the secondary ones insufficiently detailed in the process of care, so preventability of the death itself could not be assessed.

Interpretations and implications—The New Zealand Quality of Healthcare Study (NZQHS) found that AEs were associated with 12.9% of admissions, and 4.5% of AEs resulted in death. The Harvard Medical Practice Study (HMPS)⁵ produced a rate of AE of 3.7%; and of these AEs, 13.6% were associated with death. The Utah and Colorado Study (UTCOS)⁶ showed death rates of 6.6% of all AEs reported and 8.8% of all negligent AEs identified. The Quality of Australian Health Care Study² showed a death rate of 4.9%; 69.6% of these deaths were judged to be highly preventable and patients who died had 8.2 added bed-days compared with all AEs.

In these studies, and ours, data acquisition was carried out in a comparable manner (although there were some methodological differences), so these are the studies with which we can most accurately compare our results.

None of these previous reports have included critical analysis of the deaths, so raw numbers have been used to make extrapolations of the impact of adverse healthcare

and death. Extrapolating from the results of the HMPS to the whole of New York State for 1984, the conclusion was that 13451 people would have died, at least in part from an AE. The mortality rate, where death was the outcome of a highly preventable AE, extrapolated from UTCOS^{4,6} was 0.13% or 1.3 per 1000 admissions; this compares with the NZQHS rate of 0.22% (15/6579) or 2.2 per 1000 admissions.

Controversies and directions—We have attempted to estimate the number of years of life lost by the patients where we identified an association between the AE and death. For this estimate, we used the expected life span of each person.

Group A patients were much older than those patients suffering AEs overall (75.6 versus 51.5 years), and many had significant pathology or physiological compromise. Without taking their presenting disease or comorbidities into account, they lost an average of 11.8 years of life.

Group B patients are distinguished by being younger (57.9 years) than all others who died, although they were still older than the average person suffering an AE; their average loss of life was 25.0 years.

However, this assessment over-estimates the number of years lost by this particular group of patients; we are applying normal life-expectancy tables to a group of patients, most of whom already had serious disease along with comorbidities¹² which would have markedly reduced their years of life available. We have been unable to find a method that would provide an appropriate comparison of life expectancy for our cases.

The Institute of Medicine Report⁴ extrapolating from the UTCOS⁶ study of AEs, claimed that at least 44,000 Americans (1.3 per 1000 admissions) die each year as a result of medical error; this would make medical misadventure the eighth most important cause of death in the United States.

McDonald et al⁷ questioned the use of this methodology to make such extrapolations, describing the review methodology as observational and not appropriate for defining cause and effect. They concluded that the estimates of rates of death from medical errors—which were based on the methods of review used in our study—were misleading and exaggerated. We agree with his view. These cautions were countered by Leape¹³ in the same issue of *JAMA*, where he claimed that the numbers had never been seriously called into question before. In fact, there has never been a critical analysis of the causal association of death to AE in the studies reported.

Hayward and Hofer⁸ set out to answer the question of whether the implicit review tool gave an overestimate of the frequency of preventable deaths. They were particularly concerned to identify how much the death would have been preventable in the absence of an AE. Their 14 trained and carefully controlled reviewers conducted 383 reviews of 111 deaths (excluding expected deaths) sampled from VA hospitals in 1995–6. Even in this situation, there was poor inter-reviewer reliability as to preventability. The final conclusion was that only 6% of those dying would have likely survived to discharge if given optimal care, and only 0.5% of those 111 would have been expected to survive in good cognitive health for 3 months or more. Hayward and Hofer believe that, while the implicit review tool probably underestimates the occurrence of AEs, it almost certainly overestimates their consequences. Our conclusion is in concordance with his view.

In New Zealand, Tobias and Turley¹⁴ have estimated (on the basis of NZQHS findings) that about 1500 deaths per year are due to in-hospital AEs of any degree of preventability, hence making AEs the 11th most important cause of death; however, our re-analysis would reduce that estimated rate by perhaps a third.

Conclusion

Of the total reviewed population of 6579, 118 patients died; and of 850 patients suffering an AE, 38 died. Based on the American AE definition, there were 30 deaths among 659 AEs. However, only 19 of these 30 deaths (19/6579 or 2.8 per 1000 of the reviewed population) were causally related to the AEs; 63% of the raw number reported.

Where the AE itself was considered to be highly preventable, 3.4% (9/267) or 1.3 per 1000 reviewed admissions were causally related to death. It is likely that a similar re-analysis of the deaths in previous studies would reduce the number (from which extrapolations of the fatal consequences of healthcare have been made) by about a third according to our estimates. Moreover, the retrospective review process (used to determine AE rate) may underestimate the frequency of AEs, but conversely may overestimate their consequences.

Author information: Robin Briant, Formerly Clinical Director (NZQHS), Centre for Health Services Research and Policy, School of Population Health, Faculty of Medical and Health Sciences; John Buchanan, Associate Professor, Epidemiology and Biostatistics Section, School of Population Health, Faculty of Medical and Health Sciences; Roy Lay-Yee, Research Fellow, Department of Sociology, Faculty of Arts; Peter Davis, Professor, Department of Sociology, Faculty of Arts; University of Auckland, Auckland

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Correspondence: Associate Professor John Buchanan, Epidemiology and Biostatistics Section, School of Population Health, Faculty of Medical and Health Sciences, University of Auckland, Private Bag 92019, Auckland. Fax: (09) 373 7503; email: j.buchanan@auckland.ac.nz

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