

SCORTEN: A Severity-of-Illness Score for Toxic Epidermal Necrolysis

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The mortality of toxic epidermal necrolysis is about 30%. Our purpose was to develop and validate a specific severity-of-illness score for cases of toxic epidermal necrolysis admitted to a specialized unit and to compare it with the Simplified Acute Physiology Score and a burn scoring system. A sample of 165 patients was used to develop the toxic epidermal necrolysis-specific severity-of-illness score and evaluate the other scores, a sample of 75 for validation. Model development used logistic regression equations that were translated into probability of hospital mortality; validation used measures of calibration and discrimination. We identified seven independent risk factors for death and constituted the toxic epidermal necrolysis-specific severity-of-illness score: age above 40 y, malignancy, tachycardia above 120 per min, initial percentage of epidermal detachment above 10%, serum urea above 10 mmol per liter, serum glucose above 14 mmol per liter, and bicarbonate below 20 mmol per liter. For each toxic epidermal necrolysis-specific severity-of-illness score point

the odds ratio was 3.45 (confidence interval 2.26–5.25). Probability of death was: $P(\text{death}) = e^{\text{logit}} / 1 + e^{\text{logit}}$ with $\text{logit} = -4.448 + 1.237$ (toxic epidermal necrolysis-specific severity-of-illness score). Calibration demonstrated excellent agreement between expected (19.6%) and actual (20%) mortality; discrimination was also excellent with a receiver operating characteristic area of 82%. The Simplified Acute Physiology Score and the burn score were also associated with mortality. The discriminatory powers were poorer (receiver operating characteristic area: 72 and 75%) and calibration of the Simplified Acute Physiology Score indicated a poor agreement between expected (9.1%) and actual (26.7%) mortality. This study demonstrates that the risk of death of toxic epidermal necrolysis patients can be accurately predicted by the toxic epidermal necrolysis-specific severity-of-illness score. The Simplified Acute Physiology Score and burn score appear to be less adequate. *Key words: hospital mortality/logistic regression model/patient outcome assessment. J Invest Dermatol 115:149–153, 2000*

Toxic epidermal necrolysis (TEN) is a rare drug-related acute life-threatening condition. Apoptosis of cells causes erosions of the mucous membranes, extensive detachment of the epidermis, and severe constitutional symptoms (Roujeau and Stern, 1994). Stevens–Johnson syndrome (SJS) and TEN are variants within a continuous spectrum, milder forms are known as SJS or SJS/TEN overlap (Bastuji-Garin *et al*, 1993). This classification is based on the higher percentage of denuded skin; cases with the most extensive skin detachment are associated with the poorer prognosis, with a 30% death rate (Roujeau and Stern, 1994).

Three prognosis factors have been previously highlighted: age, maximal percentage of body surface area (BSA) detachment, and serum urea nitrogen level (Revuz *et al*, 1987). Furthermore, TEN-associated bronchial epithelial necrosis has been demonstrated prospectively to be a major cause of death (Lebargy *et al*, 1997).

Recently, prompt withdrawal of the culprit drug has been showed to decrease the risk of death (Garcia-Doval *et al*, 2000).

In intensive care unit (ICU), several severity-of-illness scores have been proposed to classify patients according to their vital prognosis (Lemeshow and Le Gall, 1994). The Simplified Acute Physiology Score (SAPS II), which is composed of 15 weighted variables, was assessed on a large sample of ICU patients excluding burned and cardiac patients (Le Gall *et al*, 1993). This score is currently used in the ICU to estimate the probability of hospital mortality; however, the usefulness and the discriminatory power of this score in the subset of TEN patients have not been studied to date. Specific scores have been developed in burn units, on the arguments that general scores were not adapted to burned patients. A classical commonly used score, calculated the percent likelihood of mortality as the patient's age in years plus the percent of the BSA that was burned (Zawacki *et al*, 1979); however, the usefulness of this simple score in predicting the outcome of TEN patients has not been addressed. It could be assumed that general scores are not adapted to TEN patients as well; a specific severity-of-illness score may be helpful to evaluate the efficiency of medical interventions in such patients.

The purpose of our study was to develop a TEN-specific severity-of-illness score (SCORTEN) based on a minimal set of well-defined variables, and prospectively to assess its performance by using measures of calibration and discrimination. Although

Manuscript received January 31, 2000; revised May 8, 2000; accepted for publication May 25, 2000.

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Abbreviations: TEN, toxic epidermal necrolysis; SJS, Stevens–Johnson syndrome; SAPS, Simplified Acute Physiology Score.

based on sophisticated statistical methods, our goal was to develop a scoring system that was as simple as possible and easy to use at the bedside. In addition, the SCORTEN model is a logistic regression equation that can be used to translate the score into a probability of mortality. The second purpose was to evaluate the SAPS II and a burn scoring system in these patients.

MATERIALS AND METHODS

Patients Two databases were used for this study, the first one concerned patients admitted to our dermatology ICU (Hôpital Henri-Mondor, Université Paris XII, Créteil, France) from July 1979 to December 1993. This database was used to develop the SCORTEN and to determine the usefulness of the SAPS II and burn scores for TEN patients. The second one, concerned patients admitted from January 1994 to December 1998 and was used to validate the SCORTEN. For the two databases, patients were eligible if the discharge diagnosis was SJS, SJS/TEN overlap, or TEN according to a recent classification (Bastuji-Garin *et al*, 1993). Patients were classified, respectively, as having SJS, SJS/TEN overlap syndrome, and TEN, if the maximal percentage of denuded skin was, respectively, below 10%, between 10 and 30% and above 30%. Diagnosis of staphylococcal scalded skin syndrome was eliminated by a skin biopsy. Patients included in a therapeutic trial ($n = 15$) (Wolkenstein *et al*, 1998) were not included in the study. Data analyzed were those recorded during the first 24 h in the dermatology ICU and were abstracted from medical charts. The end point was outcome at discharge from the hospital.

Clinical and biologic data Data collected included the percentage of BSA detachment (sum of detached and detachable epidermis) calculated within 24 h after admission, the clinical and biologic variables of the SAPS II which are listed in **Table I**, and other potential prognosis factors. As required to calculate the SAPS II, variables have been recorded as the worst value in the first 24 h period in our dermatology ICU. We took all variables

recorded in the SAPS II as categorical variables, and used for each the normal ranges as defined in this score. The cut-off point used to determine the high serum glucose level, which is not included in the SAPS II score, was the SAPS I cut-off point (Le Gall *et al*, 1984). To define the aspartate and alanine aminotransferase and abnormal phosphorus levels that are often observed in TEN patients, the level cut-off points chosen were those usually considered to define the normal ranges of the laboratory. As for burned patients, the arterial oxygen pressure was considered abnormal for a value below 70 mmHg (Zawacki *et al*, 1979). Acquired immune deficiency syndrome, which is not usually associated with a poorer hospital prognostic among TEN patients, was separated from hematologic malignancy and metastatic cancer for the analysis of individual parameters. If a variable was not measured for a patient, it was assumed to be within the range of normal. For each patient, the SAPS II and the classical score used for burned patients (percentage of BSA involved on admission plus age) were calculated.

Analysis

Model development All variables of patients who died were first compared with those of survivors by using univariate analyses (χ^2 square and Fisher exact test when required). Odds ratios were estimated with their 95% confidence intervals. Then, logistic regression techniques were used for model development in order to obtain a specific score and an equation for the risk of hospital death. Two by two analyses were also used to assess interaction and confusion by fitting multiplicative models. Variables emerging with a possible prognostic value ($p < 0.20$) were then entered simultaneously into multiple logistic regression models (BMDP programs). Independent prognosis factors emerging from these logistic regression models were then used to build the SCORTEN by summing the number of parameters with abnormal values. To obtain the probability of hospital mortality, the relevant model variables were multiplied by the coefficients from the logistic regression equation, and the resulting sum across all the

Table I. Univariate analysis of suspected prognosis factors of TEN in the development sample (165 patients)

	Dead patients (n=44) Abnormal values		Survivors (n=121) Abnormal values		Odds ratio (95% CI) ^a	p-value
	Percent	Numbers	Percent	Numbers		
SAPS II variables						
Age (≥ 40 y old)	72.7	(32/44)	37.2	(47/121)	4.5 (2.1–9.7)	< 0.001
Heart rate (≥ 120 per min) ^b	72.7	(32/44)	47.9	(58/121)	2.9 (1.4–6.2)	< 0.01
Systolic blood pressure (< 100 or > 200 mmHg) ^c	7.1	(3/42)	0	(0/119)	–	0.02
Temperature (> 39°C)	81.4	(35/43)	66.7	(80/120)	2.2 (0.9–5.2)	0.06
Oliguria (< 1000 ml/24 h)	24.4	(10/41)	17.7	(20/113)	1.5 (0.6–3.5)	0.35
Serum urea level (> 10 mmol per liter)	37.2	(16/43)	10.9	(13/119)	4.8 (2.1–11.3)	< 0.001
White blood cells count (< 1.0 or > 20.0 $\times 10^3$ per mm ³) ^c	6.8	(3/44)	0	(0/121)	–	0.18
Serum potassium level (< 3.0 or > 5.0 mmol per liter) ^c	20.9	(9/43)	5.9	(7/118)	4.2 (1.4–12.2)	0.01
Serum sodium level (< 125 or > 145 mmol per liter) ^c	11.6	(5/43)	3.4	(4/118)	3.8 (0.9–14.8)	0.06
Serum bicarbonate level (< 20 mmol per liter)	22.7	(10/44)	5.0	(6/121)	5.6 (1.9–16.8)	< 0.01
Bilirubin level (≥ 68.4 μ mol per liter)	4.5	(2/44)	2.6	(3/117)	1.8 (0.3–11.2)	0.52
Glasgow score (< 14)	2.3	(1/44)	3.3	(4/121)	0.7 (0.1–6.3)	0.73
Chronic disease ^d	20.5	(9/44)	5.0	(6/121)	4.9 (1.6–14.9)	< 0.01
Other variables						
Male gender	63.6	(28/44)	48.8	(59/121)	1.8 (0.9–3.8)	0.09
HIV seropositivity	15.9	(7/44)	24.0	(29/121)	0.6 (0.2–1.5)	0.26
BSA involved at day 1						
< 10%	34.1	(15/44)	64.5	(78/121)	1	–
10–30%	27.3	(12/44)	18.2	(22/121)	2.8 (1.2–7.0)	–
> 30%	38.6	(17/44)	17.4	(21/121)	4.2 (1.8–9.9)	< 0.01
Serum glucose level (> 14 mmol per liter)	29.5	(13/44)	5.0	(6/120)	8.0 (2.8–23.0)	< 0.001
Serum phosphorus level (< 0.8 or > 1.35 mmol per liter)	56.8	(25/44)	42.1	(51/121)	1.8 (0.9–3.7)	0.09
Alanine aminotransferase (> 40 UI)	63.6	(28/44)	52.1	(63/121)	1.7 (0.8–3.5)	0.16
Aspartate aminotransferase (> 40 UI)	72.7	(32/44)	54.5	(66/121)	2.6 (1.2–6.1)	0.02
Arterial oxygen pressure (< 70 mmHg)	36.4	(16/44)	14.0	(17/121)	3.5 (1.6–7.8)	< 0.01

^aConfidence interval; ^bNo bradycardia was observed in our series of patients; ^cFisher exact test; ^dMetastatic cancer or hematologic malignancy.

variables forms a value, called logit. The logit was then transformed into a probability of hospital mortality using the following equation: $\text{Pr}(\text{hospital mortality}) = \frac{e^{\text{logit}}}{1 + e^{\text{logit}}}$.

Model assessment Performance of this new model, including calibration and discrimination, was demonstrated in the second sample of patients. Calibration, which evaluates the degree of correspondence between the estimated probabilities of mortality produced by the model and the actual hospital mortality experience of patients, was statistically evaluated using a formal goodness-of-fit test (Lemeshow and Hosmer, 1982). The area under the receiver operating characteristic (ROC) curve was calculated in order to evaluate the discriminatory power of the SCORTEN (Hanley and McNeil, 1982).

Analysis of SAPS II and burn scoring systems Mean SAPS II score of survivors was compared with those of nonsurvivors by logistic regression; calibration and discrimination of the model were then analyzed. The prediction of mortality rate used the following formula: $P(\text{death}) = \frac{e^{\text{logit}}}{1 + e^{\text{logit}}}$ [$\text{logit} = -7.7631 + 0.0737 \text{ SAPS II} + 0.9971 \ln(\text{SAPS II} + 1)$] (Le Gall *et al.*, 1993). The mean score used for burn patients was also compared between these two populations. No risk of death formula was available for this score, but a ROC curve was generated to evaluate the discriminatory power of the score.

RESULTS

Study populations The first database, used for SCORTEN development and assessment of the two other scores, included 165 patients, 78 females and 87 males. The mean age was 42.3 ± 19.8 (SD) years (range 5–88). Twenty-seven patients had SJS (16.3%), 69 overlap SJS/TEN (41.8%), and 69 TEN (41.8%). The mortality rate at discharge was 44 of 165 (26.7%). The second database, used for the SCORTEN validation, included 75 patients, 33 females and 42 males. The mean age was 46.9 ± 20.2 (SD) years (range 13–92). Thirty-three patients had SJS (45.3%), 16 overlap SJS/TEN (21.3%), and 25 TEN (33.3%). The mortality rate at discharge was 15 of 75 (20%).

Model development (SCORTEN) Table I summarizes the results of the univariate analyses of suspected prognosis factors for death. None of our patients had a surgical admission; none was ventilated during the first days. Among the 13 other parameters included in the SAPS II score, nine were associated with a greater risk of death (age, heart rate, blood pressure, temperature, serum urea, potassium, sodium, bicarbonate level, and underlying chronic disease). The odds ratios ranged from 2.2 for a high temperature level to 5.6 for a low bicarbonate level. Six of the eight other parameters not included in the SAPS II were associated, or nearly associated, with a greater risk of death: BSA involved at day 1, high serum glucose and aspartate aminotransferase levels, low arterial oxygen pressure, abnormal value of serum phosphorus, and male gender. Strong relationships but no significant interaction were observed between several parameters. In multivariate analyses, seven variables remained independent prognosis factors of death: age above 40 y, heart rate above 120 per min, cancer or hemopathy, BSA involved above 10%, serum urea level above 10 mmol per liter, serum bicarbonate level below 20 mmol per liter, and serum glucose level above 14 mmol per liter (Table II). The risk estimates of these seven parameters being similar (comprised between 2.5 and 5.3), a weight of 1 was assigned to each one. Thus, the SCORTEN was calculated for each of the patients in the development sample by summing the number of abnormal parameters. The SCORTEN was then used as the only term, along with a constant term, in a new logistic equation, resulting in a model that provided an estimate of the severity as defined by the probability of hospital mortality. This model is described by the following formula: $\text{logit} = -4.448 + 1.237(\text{SCORTEN})$. For each score point, the odds ratio was 3.45 (confidence interval from 2.26 to 5.25). Table III shows the hospital mortality observed in our model development series for up to 5 points of SCORTEN.

Assessment of the SCORTEN performance The SCORTEN was then calculated for the patients of the validation sample. The hospital mortality rate predicted by the SCORTEN

Table II. Independent prognosis factors of TEN. Multivariate analysis in the development sample (165 patients)

Variables	Odds ratio (95% CI ^a)	p-value
Age (≥ 40 y old)	2.7 (1.0–7.5)	0.05
Heart rate (≥ 120 per min)	2.7 (1.0–7.3)	0.04
Cancer/hematologic malignancy	4.4 (1.1–18.0)	0.04
BSA ^b involved at day 1		
< 10%	1	0.04
10–30%	2.9 (0.9–8.8)	
> 30%	3.3 (1.2–9.6)	
Serum urea level (> 10 mmol per liter)	2.5 (0.9–7.3)	0.09
Serum bicarbonate level (< 20 mmol per liter)	4.3 (1.1–16.0)	0.03
Serum glucose level (> 14 mmol per liter)	5.3 (1.5–18.2)	< 0.01
SCORTEN	2.45 (2.26–5.25)	$< 10^{-4}$

^aConfidence interval; ^bBSA, body surface area detached. SCORTEN represents the number of abnormal parameters among the seven independent prognosis factors (a weight of 1 was assigned to each independent parameter), odds ratio corresponds to one score points.

Table III. Mortality rates and relative risks according to the SCORTEN level (development sample of 165 patients)

SCORTEN	No. of patients	Mortality rate		Odds ratio (95% CI ^a)
		Percent	95% CI	
0–1	31	3.2	(0.1–16.7)	1
2	66	12.1	(5.4–22.5)	4.1 (0.5–35.2)
3	34	35.3	(19.8–53.5)	14.6 (2.0–138.0)
4	24	58.3	(36.6–77.9)	42.0 (4.8–367.0)
≥ 5	10	90.0	(55.5–99.8)	270.0 (15.0–487.0)

^aConfidence interval, SCORTEN represents the number of abnormal parameters among the seven independent prognosis factors (a weight of 1 was assigned to each independent parameter).

Table IV. Goodness-of-fit for the SCORTEN at admission in dermatology department for the 75 patients of the validation sample^a

Probability of death	SCORTEN	No. of survivors		No. of deceased	
		Observed	Expected	Observed	Expected
0–0.099	0–1	37	36.91	1	1.09
0.1–0.199	2	14	15.01	3	1.99
0.2–0.399	3	7	4.73	0	2.27
0.4–0.599	4	2	2.8	5	4.2
≥ 0.6	≥ 5	0	0.88	6	5.12

^aThe low value of the Hosmer–Lemeshow statistic and the corresponding high p-value indicated good agreement between observed and expected number of deaths (χ^2 -like statistic, $H^*g = 2.27$; degrees of freedom = 4; $p > 0.5$).

using the “risk of death formula” ($P(\text{death}) = \frac{e^{\text{logit}}}{1 + e^{\text{logit}}}$ [$\text{logit} = -4.448 + 1.237(\text{SCORTEN})$]) was 19.6%. When the Hosmer–Lemeshow goodness-of-fit test was used to quantitate the overall fit of the model to the data (Table IV), the high p-value indicated an excellent agreement (calibration) between the observed (20%) and expected (19.6%) number of deaths ($H^*g = 2.27$, d.f. = 4; $p > 0.50$). The ROC curve generated,

revealed a good discriminatory power of the SCORTEN (area under the ROC curve = 0.82 ± 0.34 [SD]).

Assessment of SAPS II and burn scoring in TEN population The SAPS II was 21.1 ± 10.2 (SD) in survivors and 31.1 ± 11.8 (SD) in nonsurvivors ($p < 0.0001$). The relative risk corresponding to one score point was 1.09 with a 95% confidence interval from 1.05 to 1.13. The hospital mortality rate predicted by the SAPS II score was 9.1%, the low p -value of the Hosmer–Lemeshow goodness-of-fit indicated a poor agreement between the observed (26.7%) and expected (9.1%) mortality ($H^*g = 96.2$, d.f. = 3; $p < 0.001$). The SAPS II scoring system appeared to be less adequate than SCORTEN in distinguishing the patients who survived from those patients who died (area under the ROC curve: $0.72 \pm (0.52$ SD)). The burn score was significantly higher in dead patients than in those who survived (82.2 ± 30.1 versus 56.4 ± 23.2 ; $p < 0.0001$). Similarly to the SAPS II, the ROC curve generated revealed a rather poor discriminatory power with an area under the ROC curve of $0.75 (\pm 0.58$ SD).

DISCUSSION

TEN is more than an acute skin failure; all organs can be involved leading to a potentially life-threatening process. The total mortality rates observed in our series (respectively, 26.7 and 20%) were consistent with those previously reported, from less than 5% in SJS, to 30% for overlap SJS/TEN and TEN (Roujeau and Stern, 1994). Our aim was to build a simple prognostic scoring system, applicable to the whole spectrum of TEN patients, and based on variables readily available on admission. In many scoring systems, each organ dysfunction, or each variable is graded, and adding the points produces a score. The selection of variables and their appropriate statistical weights are defined by analyzes on very large databases of more than 10,000 patients (Lemeshow and Le Gall, 1994). The low incidence rate of TEN (about 1 new case per million inhabitants per year) does not allow to collect such a sample. To obtain sufficient numbers we included patients over a large time period 1979–98. The medical management of patients did not change over this period and the mortality did not evolve significantly. Thus, our development database consisted of a series of 165 patients (period: 1979–93). As required, model performance was demonstrated in another sample of 75 patients (period: 1994–98), different from those used to develop the models. The validation sample could be assembled either by randomly splitting a database into two portions, by collecting the data on a new cohort of patients (Lemeshow and Le Gall, 1994), or by using bootstrap techniques. The size of our development database did not allow us to grade the values of each variable; we only considered a variable as normal or abnormal. Variables selected for univariate analyses were those included in the SAPS II, and abnormal variables commonly encountered in TEN patients. In a retrospective study based on 87 patients, three prognosis factors were identified: age, maximal percentage of BSA detachment, and serum urea nitrogen level (Revuz *et al*, 1987). Our results confirm that the initial percentage of detachment, age, and serum urea level are independent predictors of death. Among other biologic variables, only a high level of glucose remained significant in multivariate models. We found a significant association between hypoxemia and hospital death. This was anticipated because of our previous finding of an association between mortality and bronchial epithelial necrosis (Lebargy *et al*, 1997), was expected; however, strong relationships were observed between hypoxemia, abnormal heart rate, and low bicarbonate level, so that hypoxemia did not remain as a predictive factor of mortality when controlled for the other prognostic variables in logistic regression models. Delay of withdrawal of the culprit drug is a prognosis factor for TEN (Garcia-Doval *et al*, 2000). Unfortunately, in our experience this variable is often not available because of the uncertainty of the culprit drug.

Finally using logistic regression models, we identified seven independent risk factors for death: age above 40 y, presence of

malignancy, tachycardia above 120 per min, the percentage of epidermal detachment above 10% at admission, serum urea level above 10 mmol per liter, serum glucose level above 14 mmol per liter and bicarbonate level below 20 mmol per liter. Mortality was a function of the number of these risk factors; we built a simple score by adding only seven nonweighted variables, the SCORTEN. For each SCORTEN point, the odds ratio was 3.45 (confidence interval from 2.26 to 5.25). Because of an important overlapping of the confidence intervals of the seven risk estimates we assigned the same weight to all parameters. A probability of death formula was obtained, the hospital mortality was calculated using the following formula: $P(\text{death}) = e^{\text{logit}} / (1 + e^{\text{logit}})$ with $\text{logit} = -4.448 + 1.237$ (SCORTEN). This model was validated on a second sample of patients. As required, the model performance was assessed by using measures of calibration and discrimination (Lemeshow and Le Gall, 1994). The formally test calibration demonstrated an excellent agreement between expected and observed number of death. The discriminatory power was also excellent with a ROC area of 82%.

Several severity-of-illness scores (SAPS, Acute Physiology and Chronic Health Evaluation [APACHE], and Mortality Probability Models) have been proposed to classify critically ill patients according to their risk of hospital death (Lemeshow and Le Gall, 1994). We used the SAPS II in our dermatology ICU, but this score has not been validated in these specific patients. SAPS II has been developed on a large international database of ICU patients; however, the development sample excluded burns, and TEN patients have been often compared with second-degree burns. Although evaluated at one single center over a 15 y period, our results tend to confirm that SAPS II is a suboptimal score for TEN patients, and do not support its use as a prognostic score in this specific population. Although the SAPS II was significantly lower in survivors than in nonsurvivors, there was a large overlap in SAPS II scores recorded in survivors and nonsurvivors. This is illustrated by the moderate discriminatory power shown by the area under the ROC curve (72%). If the area is about 50%, the model is performing no better than the toss of a coin. A ROC area of about 70% is not satisfying (Lemeshow and Le Gall, 1994). In addition, the large difference between observed and expected mortality across all the strata in the validation sample suggests that the model does not correctly reflect the outcome of patients.

The score proposed for burns had a similarly moderate discriminatory power as the SAPS II (area under the ROC curve 75%). In addition, information to estimate the goodness-of-fit of this score is lacking in the literature. In burn units, this score adding age in years plus the percentage of burned BSA was used for triage; this formula is easy to keep in memory (Ryan *et al*, 1998). This score, however, became obsolete because of the improvement in survival rates in major burn centers (Tompkins *et al*, 1986). Thus, complex formulas were developed requiring more sophisticated clinical variables. In a recent study, three risk factors of death were identified in burned patients: age greater than 60 y, more than 40% of BSA burned and inhalation injury (Ryan *et al*, 1998). This score is simple, but could not be applied to TEN patients. Patients older than 60, with a detachment of more than 40% BSA, and with bronchial involvement were rare in our series.

Although such probability models cannot be used reliably to predict outcome in individual patients, the information provided by probability models could play a useful part for TEN patients, as for other ICU patients. A probability of mortality of 0.40 means that approximately 40 of 100 patients with this probability would be expected to die. One cannot, however, say whether any specific, individual patient will be one of the 40 such patients who may die or one of the 60 such patients who may live. Clinicians, however, often have to discuss a patient's prognosis with family members or other medical personnel; a probability of hospital mortality is an objective assessment that can provide an additional piece of useful information in these situations. Furthermore, the introduction of a new treatment is usually preceded by the demonstration of its effectiveness in a controlled trial. An important aspect of the

conduct of such trials is the ability to define and control for the severity-of-illness of patients being studied.

Our study demonstrates that the severity-of-illness and the risk of death of patients with TEN and related disorders can be accurately predicted at admission in a referral unit by the SCORTEN. Because the patients are referred to our unit a mean of 2 d after the onset of the disease, it remains to evaluate if the SCORTEN also accurately predict the outcome when calculated on admission in primary care centers.

We are indebted to Christian Brun-Buisson and Isabelle Durand-Zaleski for their helpful review of the manuscript

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