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INFECTION CONTROL

LETTERS TO THE EDITOR

Published Data Questioned

To the Editor:

In Hegger et al's report, "Transient and resident microflora of burn personnel and its influence on burn wound sepsis," the authors state that auto-contamination via the urinary tract was responsible for 27.6% of the burn wound sepsis. If 29 patients with wound sepsis were studied and seven had the infecting organism in their urinary tract, the incidence is only 24.1%.

Secondly, Table 3 lists 27.6% as the incidence for the gastrointestinal tract. Unless these patients have cloacas, they are not the same tract. Which is it?

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Dr. Heggers, author of the article in question, was invited to respond to Ms. Scheidt's comments.

I appreciate Ms. Scheidt's comments concerning our results on auto-contamination.

Apparently, in my haste to return the galleys to the publisher, I failed to realize that an incongruity existed. After careful review of our data, I found that eight patients instead of seven out of 29 had the infecting organism. Consequently, the percentage incidence is correct. With regards to the term gastrointestinal tract, your observations are correct. It was an oversight on *our* editorial review; the correct term for Table 3 should be Urinary Tract instead of gastrointestinal.

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Catalytic Models in Hospital Epidemiology

To the Editor:

The interesting study by Chavigny and Fischer in the January-February 1983 issue of *Infection Control* demonstrates a relatively simple sampling strategy for studying the epidemiology of hospital infection.[1] A different approach of their data may result in more quantitative conclusions, especially regarding the rates of nosocomial infections in relation to the length of hospital stay (LOS).

By applying a catalytic model, as originally employed by Muench[2] for cross-sectional (point-prevalence) surveys to their data, a force of infection may be calculated. The force of infection is expressed as "effective contacts" per patient per time unit. An effective contact is defined as a contact that would lead to an infection in a susceptible (ie, previously not infected) person. According to Muench, the application of the catalytic model is based on a set of assumptions. These are represented here with slight modifications and additions to accommodate the above-mentioned survey. These assumptions include:

a. a population entirely susceptible at the start (ie, at admission)
b. a constant force of infection, measured in number of "effective contacts" per patient per time unit, no matter how complex may be the events leading up to these contacts.
c. evidence that infection has taken place, allowing for an estimate of the rate of infected patients (y) at any time (t) (ie, in this study, at the end of hospital stay)
d. all individuals sampled have spent their entire stay in the community (ie, in the hospital)
e. forces of infection have not varied greatly over a fairly long period, long enough to include the whole period of stay of all individuals entered in the study
f. mortality due to the infection is negligible; for the present study this should be read as: LOS is not greatly influenced by the infection.
g. evidence of exposure is definite and remains so until the end of the observation period.

Most of these assumptions (especially c and g) seem plausible for the hospital infections and sampling strategy under discussion. A possible exception are b and f (discussed below).

First, we will try to apply the catalytic model based on these assumptions. The model predicts that the relation between the rate of cases/patients

TABLE

ACTUAL RATES (Y) OF CASES OF HOSPITAL INFECTION COMPARED TO PREDICTED RATES*

Patient Group	Length of Stay (days)	1 (days)	y	y′	a′
I	1 - 9	5	.085	.080	.0262
II	10 - 18	14	.261	.263	.0244
III	19 - 27	23	.405	.410	.0243
IV	28 - 36	32	.530	.527	.0244
**	37 - 45	41	—	.621	—
V	>36	47.2†	.675	—	—

*Calculated as $y' = 1 - e^{-0.0246(l - 1.61)}$ *l* is mean length of stay. *a′* is the infection force as calculated for each patient group separately according to Formula 3, using 1.61 as incubation period.

Data according to Chavigny and Fischer:[1]

**hypothetical patient group

†calculated mean length of stay

exposed at any time (t) and time will be:

Formula 1:

$$y=1-e^{-at}$$

where y=rate of positive cases/number exposed,
e=base of natural logarithms,
a=infection force,
t=time of exposure

In Chavigny and Fischer's study the time of exposure is equal to LOS, after subtraction of the incubation period of hospital infections.[1] Though this incubation time may vary for different kinds of hospital infections, as a first approximation we will represent it by one value (i):

Formula 2:

$$y=1-e^{-a(l-i)}$$

where l=LOS
i=incubation period.

By weighted non-linear regression the infection force (a) and the incubation period (i) may be determined. A good approximation will be obtained by simple linear regression; for this purpose formula 2 is changed to:

Formula 3:

$$\ln(1-y)=-al+ai$$

For l, the mean of the period of hospital stay in each group was substituted. This is permissible, provided that the distribution of LOS in each group is approximately homogenous. In this way an infection force (a) of 0.0246 (effective contacts per patient day) and an incubation period of 1.61 days were calculated from the data of Chavigny and Fischer ($r^2=0.9994$, $p<0.001$). For each patient group the rates expected (according to this calculation) are represented in the table. The matching curve is represented in the figure. Separate values of a (a' in the table) were obtained from the data of each patient group by substituting the LOS, the rate and the incubation period (1.61 days) in Formula 3. These are not very different from the overall value of (a).

A "half-time" of about 29.8 days can be calculated: one-half of the patients hospitalized for that period would have suffered from one or more hospital infections.

"The success of a model depends almost entirely on whether those factors which were included turn out to be those really essential to the explanation."[2]

The value of 1.61 days for the incubation period is in agreement with expectation; for the majority of hospital infections (urinary tract and respiratory infections) it is generally thought to be about 24 hours. This and the good fit of the catalytic model suggest that the assumptions, on which the model is based, are important for hospital epidemiology.

The assumption of a constant infection force (a) during the period preceding the sampling is essential to the application of the catalytic model in a point-prevalence study. However, in Chavigny and Fischer's study the condition of the patient (case or noncase) was determined at the end of hospital stay.[1] Therefore, the relation does not necessarily indicate that the frequency of effective contacts is constant during hospital stay. The results of these calculations, however, indicate that in the hospitals in the study the number of effective contacts is proportional to the length of stay.[1]

Generally, hospital infections increase the length of hospitalization. This might invalidate assumption (f). Due to the sampling strategy, this might lead to bias; one would expect that patients with hospital infections would be overrepresented in longer-stay groups. However, most if not all of the increased rate in these patient groups seems to be accounted for by the catalytic model. This apparent contradiction may be due to the small proportion of all hospital infections that causes significantly lengthened hospitalization.[3]

The force of infection (a) expressed as "effective contacts" per patient day is comparable to the rate of infections per 1,000 patient days.[4] The main differences are that the catalytic model enables calculation from the rate of cases per patients admitted, rather than from the rate of infections, and

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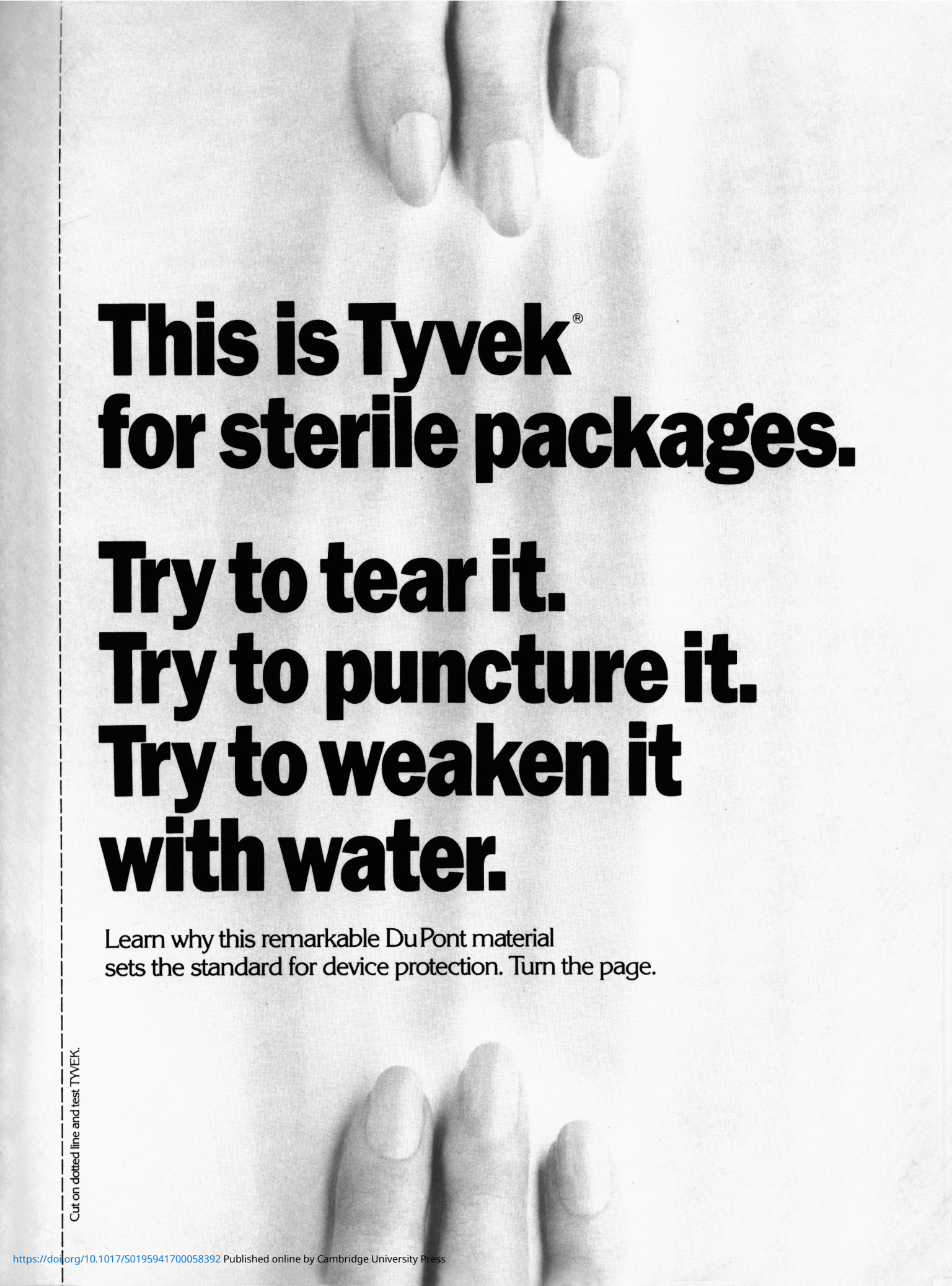

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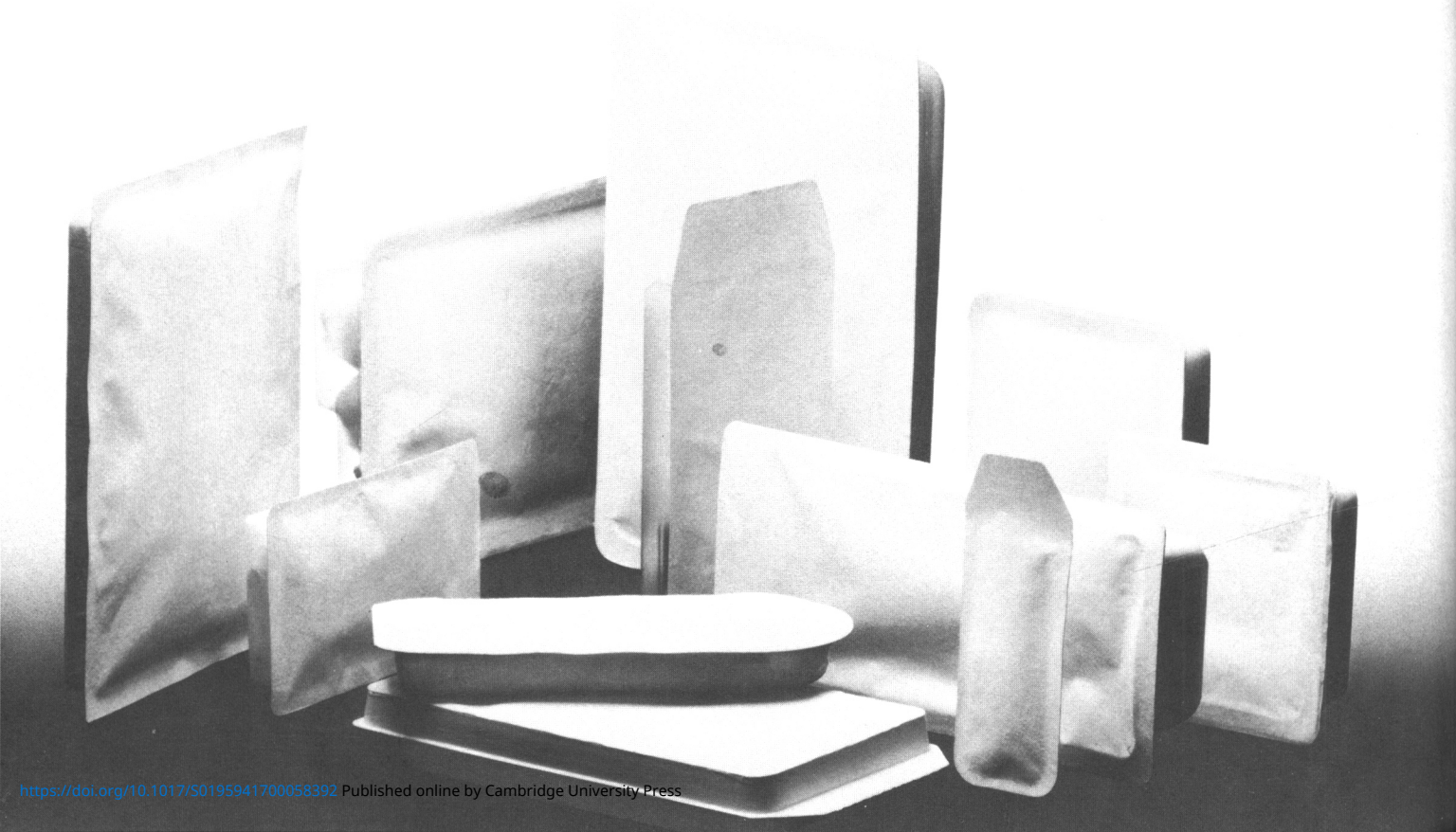

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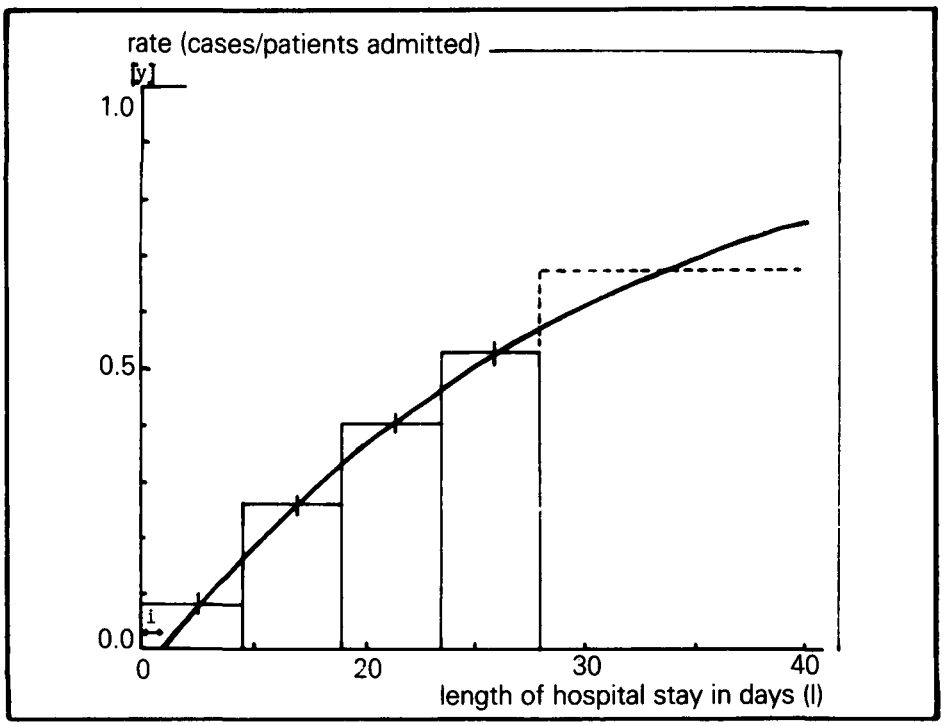

Figure. *Relation between the rate of cases of hospital infection (y) and length of hospital stay (l).* The curve $y=1-e^{-0.0246(l-i)}$ is drawn. i is the incubation period according to linear regression analysis (1.61 days).

that it allows for an incubation period. An advantage is that the force of infection may be calculated both in incidence and in prevalence surveys. The results may be different, depending on how well reality corresponds with the underlying assumptions of the model.

The contribution to the total force of infection of different kinds of nosocomial infections may be analyzed using the same model. One would expect that the catalytic model is not applicable in all instances (for example, postoperative wound infections).

We conclude that in the hospitals of Chavigny and Fischer's study the catalytic model may be used to describe the epidemiology of hospital infections.[1] Once the length of the incubation period is established, it seems possible to get a fair estimate of the "force of infection" by determining the number of infected persons in relatively small samples of patients (see the calculation of a'). Further evaluation in these and other hospitals is needed to evaluate the possible use of this parameter.

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Dr. Chavigny was invited to respond to A.J.A. van Griethuysen's comments.

Van Griethuysen's suggestion to calculate the "force of infection" from data collected from a fixed cohort with varying lengths of stay as described in the original article "Nosocomial infection in a high risk cohort: An illustration of a sampling method," presents interesting challenges.[1] In response, three issues will be raised: first, the effects of definitions and calculations in applying a catalytic formula; second, the appropriateness of using the type of analysis on data collected by this particular sampling method; and third, the implications of the suggested method of analysis for infection control practice.

A catalytic model originally applied by Muench is suggested as a method of estimating the force of infection, defined as effective contacts per patient per time unit. By changing the original formula to a simple linear regression equation, a good approximation of the infection force, "a," and the incubation period, "i," are determined. The formula for the catalytic model has been restated by Kleinbaum and Kupper[2] as follows:

$$CI \approx I - e^{-(ID)\ (t''-t')}$$

where

CI = Cumulative Incidence (number of cases of nosocomial infection divided by the population at risk); and

ID = Incidence Density rate (the number of nosocomial infection case "inceptions" over a time period (t, $t+\Delta t$) divided by the integral of the specified period during which the population at risk was followed; $(t''-t')$ is the time period over which it is reasonable to suppose a constant value for $ID_{(t,t+\Delta t)}$).

The formula can be adjusted to nearly coincide with van Griethuysen's nomenclature as follows:

$$\text{Equation (1): } y=1-e^{-(ID)\cdot\Delta t}$$

In other words, the catalytic model is a statement of the relationship between cumulative incidence ("CI" or "y") rates used in the original article and incidence density ("ID" or "a"[3]) rates of cases of infection per time in days. Van Griethuysen suggests the use of this method to produce "more quantitative conclusions" for the study; however, the use of ID rates for infection control practice is important and van Griethuysen raises a question of general interest to practice.

The original data has, in fact, been quantified through a (modified) life table analysis (Figure 1).

Figure 1 shows an (extrapolated) average population infected at 30 days of not more than 40%. The author of the letter computes > 50% infected population at 29.8 days. In addition, incidence density rates for each hospital are equal to 2.1 (Hospital A) and 1.52 (Hospital B) per 100 days, for a total rate in both hospitals of 1.76 per