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Application of Process Models in Assessment Psychology: Potential Assets and Challenges

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The relevance of quantitative models of probabilistic processes to clinical phenomena that unfold over time is discussed. Examples of such phenomena include remission processes, accumulation of objective cost and subjective stress of treatment delay, and psychopathology-related deviations in cognitive operations. Provision is made for time as a continuous or discrete variable. The discourse is developed with the use of a representative mathematical model. Extraction of information on time-related aspects of clinical transactions is described. The presentation is extended to indicate how process models can be used with established assessment methods. Challenges to the application of process models in clinical-assessment settings, and potential avenues of resolution, are put forth. It is contended that stochastic quantitative models may represent the premier analytic tack to capturing genuinely dynamic features of clinical processes.

Process models potentially are applicable to a considerable array of problems facing assessment psychologists. The type of information it is possible to obtain, and hurdles to obtaining it, depend in part on whether one's context of usage involves "basic issues of clinical science," or "immediate practical application." Basic issues of clinical science, for example, may entail dynamic aspects of a particular disorder—the course of disorder over time. Issues of immediate practical application may comprise the forecasting of change regarding a client or class of clients, under alternate treatment options. Other considerations affecting the potential benefits and challenges of process modeling include whether the addressed process is manifest primarily at the level of the individual, or larger aggregate, such as a group or institution. This article outlines what are seen as some of the more salient assets inherent in process-model implementation, barriers to such implementation, and possible avenues of resolution.

The scope of potential applications of process models in assessment is broad. The term *process* has multiple and divergent interpretations in the social and behavioral sciences (Ostrom, 1984, pp. 21–27): In the current context, virtually any series of events unfolding over time potentially could be described by some process model. Further, many mathematical distributions, each aligned with certain properties of various stochastic processes, have already been worked out and are readily available (e.g., Townsend & Ashby, 1983). In the current article, a sample of potential applications are presented and discussed with respect to one representative distribution, rather than introducing multiple distributions (and their accompanying formulas) and discussing how they each might pertain to a single application.

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Potential Utility of Process-Model Application

Process Models as Independent Sources of Assessment Information

Process models in their own right house information of potential value in clinical assessment. As well, they may enhance the utility of established measures and assessment methods. This section begins with information supplied by process models in and of themselves. Also, it necessarily contains more mathematical content than the remainder of this article. Potentially productive combinations with existing methods of clinical assessment then are examined. Illustrations are designed to focus and instantiate the subject matter appropriating a representative process model, the *gamma distribution.* The gamma distribution embodies, or is closely related to other prominent process-model distributions (e.g., the exponential distribution, described in the Special Section Introduction, the Poisson distribution, and the general gamma distribution; see, e.g., Townsend & Ashby, 1983, chapter 3). Also, the gamma distribution has a discrete-time analogue, the Pascal distribution (below), which is relatively uncomplicated, but can be highly useful for many problems.

Clinically relevant waiting processes. Consider a typical problem imposed by constraints of health care economics—that of delaying the delivery of needed treatment (e.g., Drummond, Stoddart, & Torrance, 1987). The "objective cost" of delay may entail increased expense and duration of treatment, owing to progression of disturbance and complications in the interim. Note that other substantive problems possibly more representa-

tive of those routinely encountered in certain clinical settings could readily be substituted for the present case in point. Although clinical psychologists increasingly are being called on to engage in treatment-delivery evaluation, other content issues may bear more directly on the reader's professional activities. For example, it may be more apropos to consider the problem of strategically implementing an assessment session in a psychiatric treatment setting vis-à-vis the time course following the initial intake of a client. Assessment information pertinent to client care may be vital shortly after admission, delay incurring progressive increase in cost of therapy (some symptoms may become increasingly intractable), and covert cost comprising patient distress. Other examples include the cost of delay in discharge following hospital admission, or recovery delay following symptom onset in an outpatient setting. Almost any clinically relevant event where the immediacy or delay of occurrence is part and parcel of the assessment scenario would qualify as illustrative material; the reader may freely customize examples accordingly.

Note further that in explicating the examples we use, specific mathematical formulas are used. Doing so allows an appreciation of the explicit and concise quantitative information available from process-model applications. We believe that to "convey a flavor of the meat constituting much of a future main course, the meat itself should be sampled." The formulas themselves do not go beyond basic algebra and elementary calculus (any special functions are explained). At the same time, we fully recognize that many readers would prefer to defer mathematical formalisms, pending more cordial introduction to a new and demanding topic. We would underscore, therefore, that the chief points regarding the nature, uses, and application challenges of process modeling can be appreciated without necessarily absorbing the respective formulas themselves. Those with an appetite for such material, however, need not go wanting.

A description of the distribution of waiting times is a crucial first step to predicting costs. To begin, the passage of time from the point where treatment is indicated is denoted t. The distribution of waiting times now is tentatively modeled as gamma, with parameters v and k. The gamma distribution depicts a dynamic process responsible for an event, such as treatment administration, syndrome relinquishment, or relapse occurrence. The process is said to unfold in k stages, with the number of stages transpiring per unit time being v. The probability of process completion, hence, event occurrence, at time t (e.g., t being latency to treatment onset) is proportional to the gamma distribution's density function $(vt)^{k-1}/(k-1)!\, v \exp(-vt)$. Further details on this and related distributions are presented in the Special Section Introduction, along with illustrative figures.

In the present instance, the parameter k stands for the number of events that need to transpire before treatment is made available to an individual in waiting. For example, there may be k other patients with similar problems ahead in the queue for treatment. In the present example, v would express the rate of treatment completion per unit time (e.g., 1.5 per hour or half day; more complex and perhaps realistic variants on the gamma distribution are discussed under the topic of *construct representation*, below).

Note that parameters v and k are associated with easily interpretable real-world events. Whereas many traditional psychometric techniques, such as factor analysis, provide a broad view by compressing data through regularities, process models provide a fine-grained analysis by allowing us to examine the mechanics that create these regularities.

As stated above, the relative probability of a given duration t of the waiting-time process is available according to the density function of the gamma distribution. Of particular interest may be the mean waiting time, which is k/v, and the variance in waiting time, which is k/v^2. Also of interest may be the probability of the waiting process terminating at or before a specific time t', which is the distribution function of the gamma distribution $F(t')$ (the distribution function too is elaborated on in the Special Section Introduction). In the present instance, this value is written as follows:

$$1 - \sum_{j=0}^{j=k-1} \frac{(vt)^j}{j!} \exp(-vt).$$

Furthermore, the probability of the process ending between time t' and t'', where $t' < t''$, appears as follows:

$$\sum_{j=0}^{k-1} 1/j!\, [(vt')^j \exp(-vt') - (vt'')^j \exp(-vt'')].$$

Other clinical phenomena may be regarded as the stochastic termination of dynamic processes. One such instance mentioned above is the process of symptom remission. For illustration, symptoms are considered to remit in succession, and to remain in remission, at least during the period under consideration.[1] It needs to be emphasized that full conformity of phenomena such as the present one to simplifying assumptions virtually is never realized in practice. It is anticipated, of course, that some reasonable approximation may be in place.

In the present instance, the parameter k stands for the number of individual presenting symptoms, and v stands for the rate at which each one is relinquished. Remission clearly is delayed either as v decreases, or as the initial number of symptoms k increases. The nature and use of such model parameters is dealt with further, under the topic headings of *Construct representation* and *Aptitude-treatment-intervention.*

We turn our attention now to phenomena that may be more veridically regarded as involving discrete rather than continuous time. Here, the continuous variable t gives way to that of the discrete variable n, representing distinct episodes, or interludes, during which process termination may take place. Discrete-variable status traditionally has been instantiated as "trials," coin tosses, etc. (sometimes designated "urn models," used in most introductory psychology statistics courses).

[1] It is possible, as well, to accommodate a simultaneous, or parallel symptom-remission process within a gamma distribution, given certain assumptions about changes in remission rates for symptoms that remain, after others subside. The issue of "model mimicking" is one receiving considerable attention in the domain of stochastic modeling of psychological processes (see, e.g., Townsend, 1990). It is discussed briefly in this article, in the section on challenges to process modeling in applied settings.

In the above example of waiting for treatment, n may relate to one's ordinal position in a sequence of appointments for treatment within a given setting, or with a certain practitioner. Now there are k other individuals ahead in the wait for treatment of a disorder of similar urgency to that of the individual in question. For the present purposes, the waiting process is considered to be over when the k other individuals have been serviced. Instead of υ, the rate of dispensing treatment, we now invoke the parameter p. This parameter represents the probability of an appointment being allocated to one of the k antedating positions in the disorder-severity group to which the present case belongs. The probability of an appointment's assignment to someone from another group in waiting, on the other hand, is q, or $1 - p$.

A discrete-variable distribution of potential value in quantifying the foregoing process is the Pascal distribution.[2] This distribution prescribes the probability of the waiting process being completed with appointment n, where $n \geq k$, as

$$\left[\frac{(n-1)!}{(k-1)!(n-k)!}\right](p)^n(1-p)^{n-k}.$$

The mean value of n on process completion is k/p, and the variance is kq/p. In addition, the probability that the kth intake—the intake moving the present individual to the front of the queue in his or her group—will occur at or before appointment n', where $n' = k, k + 1, k + 2, \ldots$, is found to be

$$1 - \sum_{j=0}^{k-1}\left[\frac{n'!}{j!(n'-j)!}\right](p)^j(1-p)^{n'-j}.$$

Moreover, the probability of moving to the front of the queue within some given interval can be calculated. For example, the probability of emerging at the front somewhere between appointments n', and $n'' + 1$, where $n' = k, k + 1, k + 2, \ldots$, and $n'' = n' + 1, n' + 2, \ldots$, appears as follows:

$$\sum_{j=0}^{k-1} p^j/j!\left[\frac{n'!}{(n'-j)!}(1-p)^{n'-j} - \frac{n''!}{(n''-j)!}(1-p)^{n''-j}\right].$$

The Pascal distribution once more can be integrated with other clinical events. Included, for example, is the termination of a course of successful treatment. Reaching a target clinical state can be envisioned as entailing a quota of k sessions, each resulting in some significant therapeutic gain, or milestone. The probability that a given session will be productive is conveyed by the parameter p. In turn, the probability that the kth requisite session will occur with session n again may be informative, along with other summary statistics enumerated above. Zucchini (in press), for example, has used this distribution to model empirical data on the number of patient visits for certain problems made to a sample of general practitioners.

Cost considerations. Identified with various waiting times presumably are associated costs. Included are costs of delaying treatment, in the case of queuing behind other patients, costs of delay in remission, and costs of prolongation of a course of treatment sessions. In other words, costs are considered to be functions of time. As functions of time, they can be viewed conjointly with waiting times themselves. Ideally, a valid cost function $C(t)$ follows some tractable trajectory across time.

Under certain circumstances, the function $C(t)$ may be conceived as being highest at the beginning of the epoch under consideration. For instance, a treatment program may be inaugurated prematurely, prior to appropriate assessment and diagnosis. In the case of an apparently psychotic patient, a suboptimal course of pharmacotherapy may be commenced. Deferring such treatment pending fuller assessment may be less deleterious than more immediate administration, given possible side effects and minimal likelihood of further deterioration of patient state in the meantime. With such a scenario in place, the path of $C(t)$ may be approximated by a negative exponential function, or $w\exp(-wt)$, where w determines the speed of decline (illustrated in Figure 1).

The expected or mean cost of waiting $E(C)$, taken across all values of t, technically is defined as

$$\int_0^\infty f(t)C(t)dt,$$

where $f(t)$ is a density function and again is proportional to the probability of process termination at time t. The variance in cost $Var(C)$ in turn is defined to be

$$\int_0^\infty f(t)[C(t)]^2dt - \left[\int_0^\infty f(t)C(t)dt\right]^2.$$

Where the waiting times are gamma distributed with parameters υ and k, and where the cost function is $w\exp(-wt)$, the value of $E(C)$ turns out to be the very simple and convenient expression

$$\frac{w\upsilon^k}{(\upsilon + w)^k}.$$

The value of $Var(C)$ also is readily available in this case, specifically as follows:

$$\frac{w^2\upsilon^k}{(\upsilon + 2w)^k} - \left[\frac{w\upsilon^k}{(\upsilon + w)^k}\right]^2.$$

It is possible that a cost function best reflects the peril of delay in completion of a process. Delay in stabilization of certain behaviors associated with schizophrenia, for example, may prove severely disruptive to basic self-maintenance functions (e.g., Neufeld, Carter, Nicholson & Vollick, in press). The cost function, therefore, may accelerate over time. Such a function is the positive exponential, $w\exp(wt)$, where w again denotes the rate of change over time (illustrated in Figure 2). In this case, $E(C)$ is equal to

[2] The Pascal distribution is also known as the binomial waiting-time distribution, and more commonly, as the negative binomial distribution with k as an integer of one or more.

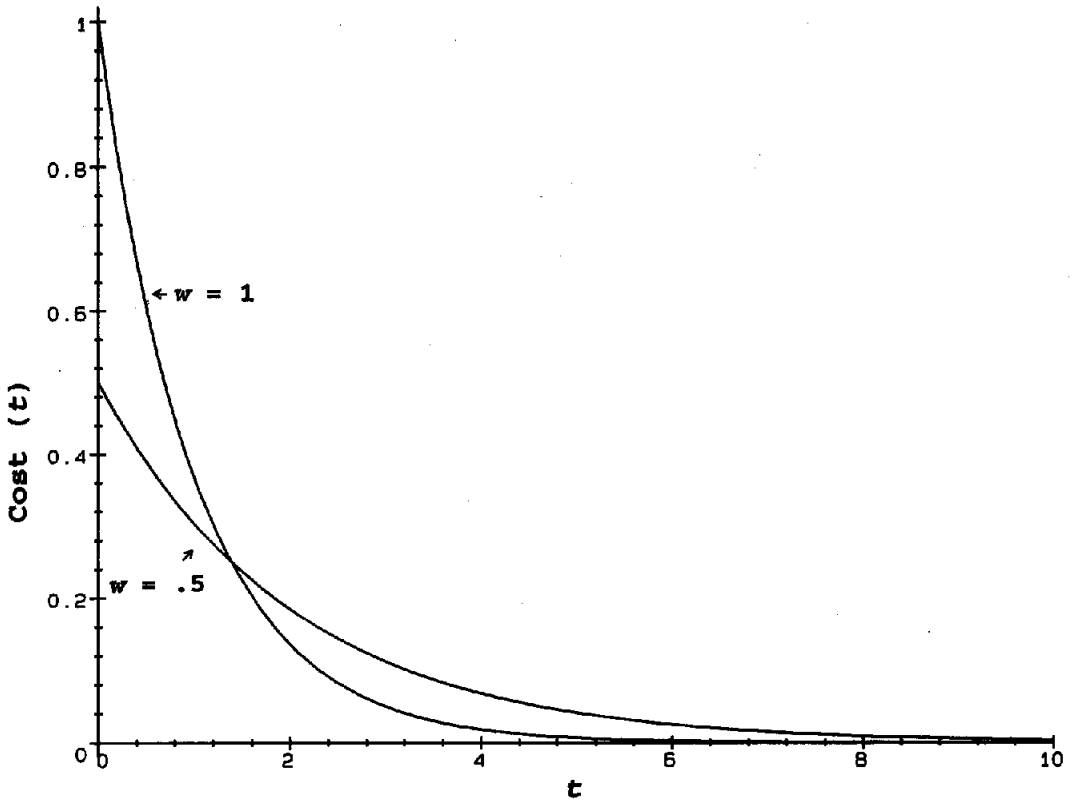

Figure 1. Negative exponential cost function, Cost $(t) = w\exp(-wt)$, for two values of w.

$$\frac{wv^k}{(v-w)^k}; \quad v > w,$$

and $Var(C)$ becomes

$$\frac{w^2v^k}{(v-2w)^k} - \left[\frac{wv^k}{(v-w)^k}\right]^2; \quad v > 2w.$$

Finally, some scenarios ostensibly call for emphasis on surges in cost associated with certain delay intervals. There may be phases where florid symptomatology is especially prominent, heightening the cost function in their wake (see, e.g., Norman & Malla, 1993). Adverse social consequences of the symptom episode are unleashed, and behavior becomes more refractory to the postponed intervention. Costs of administered treatment putatively decrease subsequently, during less behaviorally turbulent periods. In such cases, a unimodal function resembling the gamma distribution itself would appear to be in order (illustrated in Figure 3).

A cost function of time meeting these requirements, and resembling closely the above gamma distribution, is

$$\frac{(wt)^{u-1}}{\Gamma(u)} w \exp(-wt),$$

where Γ is the "gamma function."[3]

Like the earlier gamma distribution, this function possesses two parameters, these now being w and u; however, the parameter u is continuous unlike its predecessor k, which was an integer. The maximum value of the function occurs where t equals $(u-1)/w$ (see Figure 3). The parameter u may reflect cost influences of treatment delay, such as exacerbation of symptoms with the continuing stress of disorder. Accordingly, maximum cost is moved to the right on the time continuum. The opposite occurs with an increase in w. The latter parameter may be identified with suboptimal treatment selection during earlier phases, prior say to the development of stable diagnosis. Given this unimodal cost function, and waiting times that are gamma distributed with parameters v and k, $E(C)$ becomes

[3] Recall that the gamma function is the continuous-variable analogue of the factorial. Where u is an integer, $\Gamma(u) = (u-1)!$ Formally,

$$\Gamma(u) = \int_0^\infty x^{u-1}e^{-x}dx; \quad \Gamma(1) = 1.0; \quad \Gamma(0) = \infty.$$

Tables of values for the gamma function are available in standard reference sources such as Beyer (1984) or Fogiel (1980). Apropos of distributions, strictly speaking, the designation of *gamma distribution* is reserved for the case where the parameter, u, is continuous. When u is an integer (thus replaced by k, in the text's formulas), the label *Erlang distribution* is appropriate.

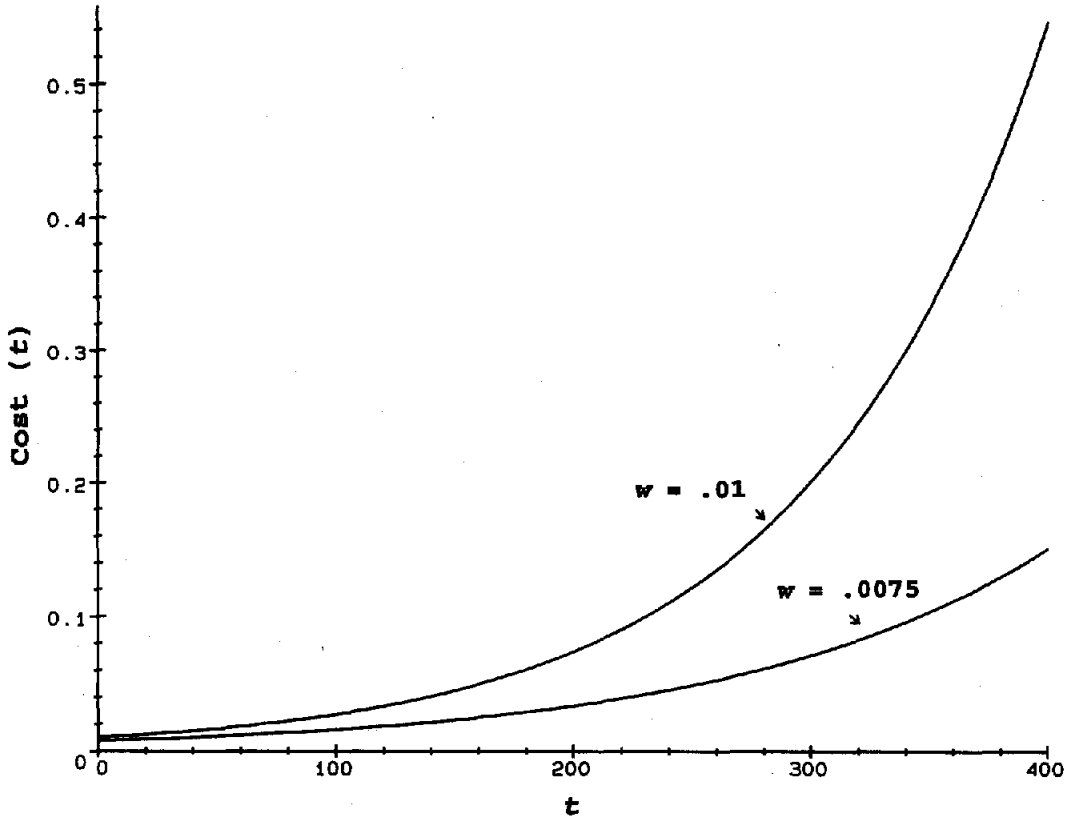

Figure 2. Positive exponential cost function, Cost $(t) = w\exp(wt)$, for two values of w.

$$\frac{v^k w^u \Gamma(k+u-1)}{(k-1)!\Gamma(u)(v+w)^{k+u-1}},$$

and $Var(C)$ is

$$\frac{v^k w^{2u} \Gamma(2u+k-2)}{(k-1)!\Gamma(u)(v+2w)^{2u+k-2}} - [E(C)]^2.$$

Note that the above combinations of cost functions and waiting times lend themselves to the stated convenient formulas for $E(C)$ and $Var(C)$. Other combinations may generate more unwieldy results, requiring numerical solutions to be performed with appropriate computer algebra programs. For example, rather than the negative exponential's density function, $w\exp(-wt)$, the following cost function may best approximate the actual expense of waiting t units of time: $w\exp[-(wt)^2]$. Combined with the present gamma-distributed waiting times, succinct formula for $E(C)$ and $Var(C)$ now are not forthcoming. Numerical solutions for given parameter values, however, readily are available with the aid of computer-algebra programs, such as Waterloo Maple or MATLAB. Parameter values, in turn may be estimated for the waiting-time and cost-function distributions, separately, using procedures like those described in connection with issues of implementation in clinical-assessment settings, below.

Similar considerations apply to discrete time counterparts, such as the Pascal distribution, where the cost function this time is expressed in terms of the discrete-time variable n, that is $C(n)$. The expected or mean cost $E(C)$ is now defined as

$$\sum_{n=k}^{n=\infty} \frac{(n-1)!}{(k-1)!(n-k)!}(p)^k(1-p)^{n-k}C(n),$$

and $Var(C)$ as

$$\sum_{n=k}^{n=\infty} \frac{(n-1)!}{(k-1)!(n-k)!}(p)^k(1-p)^{n-k}[C(n)]^2 - [E(C)]^2.$$

Succinct formulas, such as those illustrated for the case of continuous time, can be more elusive here. Suffice it to say, however, that numerical solutions again are readily available.

Cost functions described above are deterministic. That is, there is a specific unwavering value of $C(t)$ or $C(n)$ identified with waiting time t, or n. In practice, however, costs at each waiting time may themselves be stochastic in nature. For each value of n, for example, there may be several possible events, each with its own probability of occurrence, and cost incurred contingent on that occurrence. To illustrate, delay in treatment by n units may signify several possible resulting states of the disorder under consideration. These states may range in the severity of personal distress and social disruption engendered, each state having an associated magnitude of cost. Each state, as well, has its own conditional probability of occurrence, given

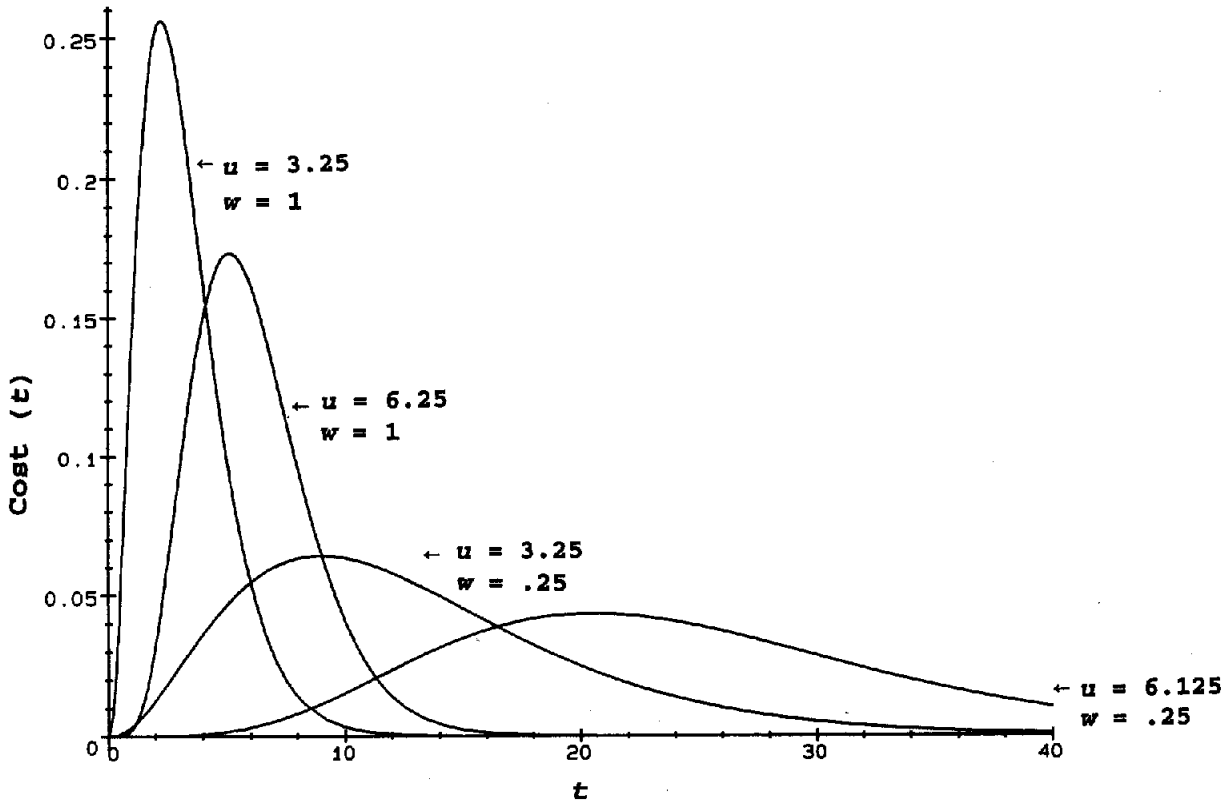

Figure 3. Gamma-defined cost function, Cost $(t) = (wt)^{u-1}/\Gamma(u)\ w\exp(-wt)$, for two values of u and two values of w.

the level of n at hand—$Pr(\text{state}|n)$. Rather than $C(n)$, the relevant term for cost computations then becomes the *conditional expected cost,* given n, or $E(C|n)$. This term takes into account the conditional probability of each candidate state, and its associated cost, by summing the respective cross-products involving $Pr(\text{state}|n)$ and the cost associated with the state, given n. With this type of extension, the computation of $E(C)$ obviously becomes more intricate; that of $Var(C)$ increases substantially in complexity (e.g., Parzen, 1962, p. 55). It must be decided, for a given problem, if the added veridicality afforded by imbuing the cost function with a stochastic element is worth the difficulties introduced by this extension. Overall, the establishment of costs in the clinical arena of course is far from being an easy matter, and it has been a longstanding subject of discussion (e.g., Drummond et al., 1987; Neufeld, 1977, chapter 6).

Further extensions of basic formulas are possible. In the case of recurrence of symptoms, for example, the parameter k may represent the number of precipitating incidents, and the parameter v may represent the rate at which these incidents occur. In the case of schizophrenia, for example, the incidents may involve emotionally charged interactions with family members. The distribution function $F(t)$ describes the probability of symptoms at or before time t. The gamma distribution would predict that symptoms would inevitably return. A simple modification (i.e., multiplying the distribution function by δ, where $\delta \leq 1.0$; Carter & Neufeld, 1996; Chechile, 1987), however, allows for the possibility that an event, such as symptom recurrence, never transpires. In this case, δ is easily interpretable as the probability of relapse and $1 - \delta$ as the probability of complete recovery.

A somewhat more complex example involves changing the assumptions underlying the chosen distribution. Consider predicting the effectiveness of interventions for depression by measuring when depressed mood recurs following treatment. Intervening events could be negative thoughts. Some therapeutic interventions, such as cognitive training, involve practice, and others, such as medication, do not. Perhaps the gamma distribution, which assumes that there is some kind of maintenance or rehearsal keeping the probability of events transpiring at a constant, would be suitable in the first case, but not in the second. The Weibull distribution (cf. Carter & Neufeld, 1998; Chechile, 1987) does not make this assumption, and is similar to gamma, except that k is set equal to one and a new parameter, β, is added. The parameter β represents the rate of increase of the probability of the critical event transpiring, given that it has not yet transpired (i.e., an increasing hazard function), and the new distribution function is $1 - \exp[-(vt)^{\beta}]$. The possibilities for similar modifications are vast. In many cases, the relevant formulas are either already available or easily computed. Other cases, such as combining multiple precipitating events with an increasing rate of probability of each, have more complex process-model counterparts (e.g., McGill & Gibbon, 1965).

To summarize, stochastic process models can contribute substantially to the conceptualization and quantification of clinically important processes. The above developments have rallied around a specific parametric model, the gamma distribution. This distribution, and its discrete-time counterpart, may have numerous applications. It is emphasized, however, that although substantive issues surrounding this exemplary distribution essentially are general, the gamma distribution is but an illustrative prototype. A goodly assortment of other well-chartered distributions are available. Their summary statistics (e.g., means and variances) and other properties are presented in highly accessible sources, such as Evans, Hastings, and Peacock (1993), and Patil and Joshi (1968). It should be reiterated as well that the substantive issues addressable by process models, presented here, instantiate a small subset of the assortment of problems that may be broached with such analyses.

Process models and the psychological cost of waiting. Potentially useful information yielded by process models can be depicted with further representative examples. In the immediate case, emphasis is placed on the psychological as opposed to objective cost of waiting for a desired or needed clinical event. A credible "nonparametric model" (i.e., "nonparametric" in the sense of not being tied to a specific distribution like gamma; see Special Section Introduction for elaboration) of the stress of waiting has been presented by Osuna (1985) and developed further by Suck and Holling (1997). Details of their developments must be left to the original sources, because of space constraints and the technical tenor of the presentations. Pertinent to the current discussion, however, are selected results of their process analysis. Specifically, the expected *psychological* cost of waiting for the desired event is the expected, or average value of the term $t\,c(t)$—that is, the average cross-product between event delay, t, and the momentary subjective value of loss incurred by t, or $c(t)$. According to these expected values, the modeled psychological cost assumes a somewhat different format from the objective cost of waiting.

To help make the above point, we considered an idealized case. The objective cost and subjective value of incurred loss are deemed to be constant across time. In other words, $C(t)$ is defined to be the constant, C, and $c(t)$ is defined to be the constant, c. In such an instance, the expected or average objective cost of waiting now reduces to C itself. However, the expected psychological cost of the wait becomes $c\mathrm{E}(t)$, where $\mathrm{E}(t)$ is the expected waiting time, or mean waiting time. Returning to the gamma distribution, the comparative values would be C in the objective case, and ck/v in the subjective case. Unlike objective cost, then, the subjective toll as averaged across all points of delay increases with the mean amount of delay, even if the subjectively appraised penalty brought on by delay does not change from one point to the next. In this way, the passage of time itself theoretically takes a psychological toll, over and above the strict assessment of benefits forfeited (cf. McFall & Townsend, 1998). Analogous expected values, in the case where n replaces t, take the form of C and ck/p. This example illustrates how counterintuitive but potentially valid results are available from the formal inferences prescribed by process-model application. The specific calculations leading to the present deductions, and associated assumptions, are available in Osuna (1985) and Suck and Holling (1997).

Random status of process-model parameters. Before leaving the domain of process models as freestanding agents of information, mention should be made of the possibility of treating parameters of a process model as variables whose values are randomly distributed in their own right. Doing so can increase model utility with only a slight increase in model complexity. Greater potential utility stems from the prospect of accommodating additional sources of variation occurring in the setting of application. Consider, for example, circumstances where the position of an individual in a queue for treatment, expressed as the gamma distribution's parameter k, may fluctuate. Prevailing values of k may be a function of local health-authority resources. As resources become more plentiful in a given geographical region, k may tend to decrease, and the opposite. The probability of k assuming a particular value may be expressed according to a distribution commonly used in this type of situation, known as the Poisson distribution. The probability of k attaining a certain value, say k', is defined by this distribution as follows:

$$Pr(k') = (m)^{k'}/k'!\exp(-m).$$

In this expression, the parameter, m, of the (simple) Poisson distribution indicates the tendency of k to assume larger values. The average value of k, given a prevailing value of m, is m itself, as is the variance of k (see, e.g., Kenny & Keeping, 1951). It may be possible to define m according to empirical variables that determine its amounts. Such variables, for example, may pertain to available health care resources. As these resources increase, m presumably decreases, and the Poisson distribution of k tends toward lower values. Consider the Poisson distribution of k for differing values of m, presented in the three-dimensional Figure 4.

Assume once again that the waiting time t in a given instance of queuing is gamma distributed. The parameters of the gamma distribution, in the instance at hand, are v and whatever value of k' is randomly bestowed on the present instance by the Poisson distribution. The expected waiting time, where the Poisson distribution with parameter m governs the obtained values of k, then would be m/v (for details, see, e.g., Ross, 1983). The corresponding expectation where the gamma distribution is replaced by its discrete-variable counterpart, the Pascal distribution, would be m/p. Rounding out this extension with considerations of cost, entertain once more the special case where the objective cost of delay is constant over t or n. Here, the expected or average cost of waiting, once more is the constant, C. Letting the subjective loss incurred by t be constant, as well, the modeled subjective cost becomes cm/v; the corresponding value in the discrete-variable case would be cm/p. (Additional background derivations and applications, pertaining to k as a Poisson-distributed random variable can be found under the topic "compound Poisson distribution," expounded, e.g., in Feller, 1966; Gibbon, 1992; or Ross, 1983.) The treatment of parameters as random variables is revisited under the discussion of individual differences, below.

Other articles in the Special Section present specific methods

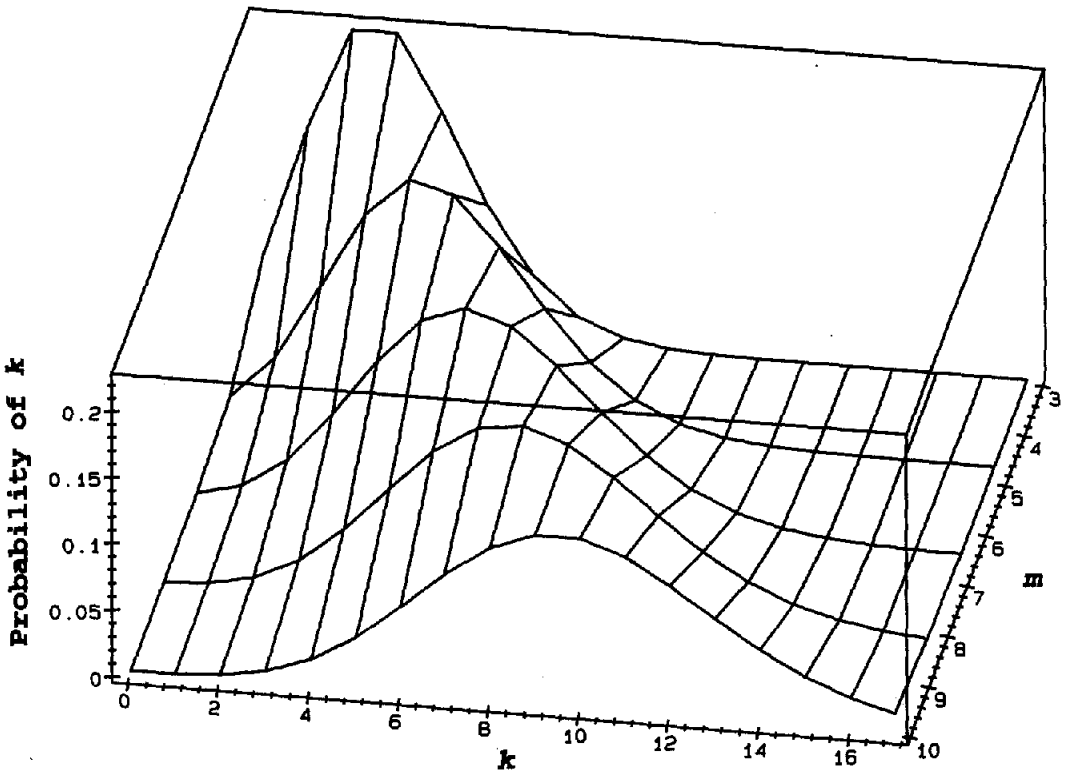

Figure 4. Poisson distribution of k for values of m ranging from 3 to 10.

for obtaining assessment-relevant information from the application of process models (see Luke & Homan, 1998; Santor & Ramsay, 1998, for continuous-variable applications, as described in the Special Section Introduction; and Batchelder, 1998, for discrete-variable applications). We turn our attention, now, to implementations where the combination of process modeling with more conventional assessment methods can mutually enhance the informational yield of each.

Process Models in Combination With Other Measurement Methods

Construct representation. An accepted dictum of technology, including measurement technology, is that "there is nothing so practical as a good theory." On this note, the value of a psychological construct is enhanced if it embraces mechanisms that are theoretically meaningful. This aspect of construct utility has been labeled in a treatise on the topic by Embretson (1983) as *construct representation.* It goes without saying that the construct validity of a measure increases with the richness of its construct representation. Commonly used measures, including those of intellectual ability and psychopathology, stand to acquire greater construct-representation value through associations with pertinent distribution properties (described in the Special Section Introduction) and parameter values of process models.

To illustrate, a psychometric measure of spatial abilities attains greater construct-representation characteristics if it predicts cogent aspects of performance on relevant cognitive tasks. These performance aspects include the ones putatively tapped through the application of process models (e.g., speed of encoding physical stimulus features, the number of stages involved in accessing relevant memory-held information, etc.). To illustrate further, a measure of depression can be made more meaningful if its scale scores are associated with stages of remission and their rate of completion, again tentatively estimated with appropriate process models. Reciprocally, the interpretation of properties and parameters rendered by process models may be fortified through associations with relevant clinical-assessment measures. For instance, the value of a process-model parameter thought to indicate speed of stimulus-feature encoding (e.g., v of the gamma distribution, where the latter legitimately models feature-encoding latency) would be expected to correlate with a psychometric measure of spatial ability subserved by efficient encoding.

Process models may also contribute to research concerning diagnostic systems. To illustrate, the relationship between child- and adult-onset schizophrenia is unclear. Comparisons of the processes underlying such similar conditions (see, e.g., Batchelder, 1998, this issue; McFall & Townsend, 1998, this issue) can supplement other diagnostic procedures. In disorders such as schizophrenia, where there are many biological correlates but not one definable marker, process models may ultimately illuminate connections between physiology and symptomatology.

Certain features of clinical disorders may be accessible primarily, or exclusively with the aid of process models. Inventory measures of psychopathology dimensions, such as paranoid versus nonparanoid forms of schizophrenia, or somatic versus affective components depression, can be correlated with such features. If so, dimensions of psychopathology addressed by the inventories secure additional construct representation through their association with dynamic properties of a syndrome. To elucidate these points and motivate the discussion, consider once again the elementary representation of symptom remission in terms of the gamma distribution.

According to the associated model, there are several steps to remission, specifically k in number, the rate of remission being v stages per unit of time. In other words, there are two model parameters. The first, k, is considered to reflect the number of protuberant symptoms presenting at intake. The second is assigned to the rate at which individual symptoms are remitted. Values of these parameters may be correlated with scales from selected inventories. Successful presaging of parameter values for individuals, or classes of individuals, endows the respective scales with added construct representation by linking them to the dynamics of recovery. Ideally, the parameter values are differentially correlated with alternate scales, affording differential prediction of the alternate parameter values.

Potential contribution to construct representation may be carried a step further, with respect to treatment effects. Process models are poised to provide useful information regarding the nature of changes occurring with adopted interventions. The locus of effects now pertains to dynamic aspects of the disorder that stand to be altered.

To illustrate, we return to the gamma distribution as a model of symptom assuagement. The administration of selected antipsychotic medication, for example, may affect the estimated rate v at which symptoms are relinquished. Conversely, the repertoire of symptoms k to be dealt with over the course of treatment may be diminished (e.g., Nicholson & Neufeld, 1993). The degree to which one or the other parameter value is altered in conjunction with the medication is apt to vary across individuals. Dominance of change in one versus the other parameter can be cast as a parametric expression of differential treatment response. A measure's construct representation once again is increased according to its association with the pattern of parameter changes.

In a similar vein, the locus of nonbiological treatment may be of interest. Therapeutic gains may be achieved during treatment sessions, as well as between sessions (possibly abetted by interpolated therapeutic cognitive exercises). Sessions tentatively are spaced at longer or shorter intervals. Allowing more or possibly less time between sessions can affect certain aspects of the course of disorder, vis-à-vis therapeutic efforts. If one now implements the Pascal distribution, the parameter p once more is aligned with the probability of significant gains taking place during a given session, and k, the number of gainful sessions mediating some target clinical state. Prolonging the time between successive sessions may be found to increase estimated values of p, tentatively because of an increase in client motivation to capitalize on therapy sessions or because of incubation of prior-session benefits. With more time for intervening exercises, on the other hand, the requisite number of scheduled therapy sessions k may be lessened, estimated values of p remaining essentially unaltered. Alternatively, estimated values of p may increase with shorter intervals between sessions as momentum or perseveration is capitalized on. Empirical prediction of treatment milestones, with reference to recovery latency and forestalling of relapse, is developed by using other distributions by Luke and Homan (1998, this issue).

In each of these instances of parameter change, duration of treatment, quantified as the total number of sessions on which the target state is achieved, decreases on average. The mechanism of reduction, however, differs in potentially significant ways. Competing mechanisms are suggested by changes in alternate features of the referent process model. Increased construct representation is available for measures predicting which feature or features are apt to change for which client or class of clients. This theme is developed further under the *Aptitude-treatment-intervention* heading, below. Meanwhile, keep in mind that the present illustration is idealized for economy of presentation. In practice, the operative model and parameters responsible for observed events may be enigmatic. Such a result is not necessarily all bad, however, a point to which we return in the section titled Challenges to Implementing Process Models in Assessment Settings, below.

Resuming with potential advantages, multifarious additional dynamic correlates plausibly add to a psychometric measure's arsenal of construct representation. The respective properties may embody unique material regarding the nature of disorder, and the nature of amelioration associated with candidate interventions. By using the ordinary gamma distribution, in the scenario above, we deemed symptoms to undergo remission in sequence; remission did not commence on a given symptom until finalization of the symptom in progress (not unlike runners taking turns in a relay; see Footnote 1). A model that instead depicts the remission process as taking place on all symptoms simultaneously might be more appropriate. The time frame of analysis arbitrarily allows the workings to be set in motion at the commencement of treatment. Although the remission process takes place on all symptoms simultaneously, the respective expirations are staggered, being distributed probabilistically across time (analogous to simultaneously kinetic billiard balls randomly falling into pockets). The speed of remission applicable to each symptom, considered individually, may be parsimoniously regarded as identical to that of each of the other symptoms undergoing the remission process in parallel. In addition, the rate of remitting may stay unchanged for symptoms remaining active as others subside. If such rates are unaffected by the total number of symptoms involved, and given one or two additional assumptions, the process is one known as "a pure death system," one commonly used to depict parallel processes in various fields of study (e.g., Ross, 1983; Townsend & Ashby, 1983, pp. 85–86). On the other hand, the speed at which continuing symptoms undergo remission may increase substantially, as other symptoms successively discontinue (known as a "pure birth system," again given some surrounding assumptions; e.g., McGill & Gibbon, 1965).

Additional variations include provision for systematic differences in rates of remission, as determined by the symptom

being addressed (e.g., Townsend & Ashby, 1983, chapter 13; Vorberg & Ulrich, 1987). It may be expected that some symptoms (e.g., schizophrenia delusions) are intrinsically less tractable than others (e.g., associated features of schizophrenia, such as depression, or anxiety). Allowance can be made also for the possibility that only a subset of symptoms needs to lapse for some target clinical state to be realized (cf. Colonius & Ellermeier, 1997; Schulz & Albert, 1976; Townsend & Colonius, 1997). Furthermore, accommodation can be made for the eventuality that the initial symptomwise speed of remission may change according to the number of symptoms that are copresent at the outset of the analyzed time period (cf. Ashby, Tein, & Balakrishnan, 1993; Townsend & Nozawa, 1995). Included in this class of developments are important advances in nonparametric or distribution-general methods for ascertaining the operation of potentially important model variations enumerated above (e.g., Townsend & Nozawa, 1995). Nonparametric methods, as used in the present context, pertain to those not tied to a particular distribution, such as the gamma distribution (see Special Section Introduction article for elaboration).

The present discussion has focused on associations of measures with features of process models expressing change in disorder over time. Associations stand to enhance the construct representation of measures according to the theoretical significance of model features involved. As indicated, among such features are those possibly amenable to administered treatment. Attention now is turned to the contribution of process models to construct representation involving measures directed to other clinically important areas. One such area is that of cognitive assessment. Productive application of process models has flourished in the field of cognitive science (see, e.g., Batchelder, 1998; McFall & Townsend, 1998, this issue). Indeed, the majority of important model advances cited above have emanated from that substantive domain. The present discussion emphasizes specifically construct representation conferred by cognitive process-model correlates.

The gamma distribution is recruited once more in the service of illustration. Note that because of its common alignment with serial-, rather than parallel-processing structures, this distribution model does not necessarily square with the apparent architecture of many cognitive mechanisms (e.g., Townsend & Ashby, 1983; see Footnote 1). Nevertheless, the gamma distribution is relatively simple, presumably is now familiar to readers of this Special Section on process models in psychological assessment, and embodies the fabric of other process models brought into play in the discussion that follows.

As stated above, a psychometric measure of selected spatial abilities would be expected to correlate, for example, with what are thought to be certain component functions. One such function may entail the efficient encoding of physical properties of everyday items (e.g., kitchen appliances, common animals, etc.) that pertain to judgments of overall item magnitude (cf. Paivio, 1975; Paivio & Harshman, 1983). Greater ability, as estimated by the test at hand, in principle could be correlated with higher speed of encoding (higher values of v), more efficiency in encoding, operationalized as fewer component stages (lower values of k), or both.

Construct representation of psychometric measures, in other words, is endowed through model features that are meaningful in their own right (e.g., v and k, above). Such model features, however, can go further in conveying construct representation if they are associated with additional clinically relevant variables. For instance, psychometrically measured thought-content disorder (related to paranoid schizophrenia delusions) has been identified with adverse changes in certain parameters of cognitive-performance models (e.g., parameters resembling k) but with integrity of other parameters of such models (e.g., parameters resembling v; Neufeld, Vollick, & Highgate, 1993). These findings, in turn, have been implemented in neighboring models of memory (Chechile, 1987), so as to draw out implications for impairment in more global aspects of information processing (e.g., multidimensional semantic, and other judgments; Carter & Neufeld, 1996; Neufeld & Williamson, 1996).

Related process-model features have been probed with respect to compromised negotiation of environmental stressors and demands (illustrations can be found in Benn & Neufeld, 1996; Morrison, Neufeld, & Lefebvre, 1988; and Neufeld & McCarty, 1994). Psychometric measures linked to model features elucidating ecologically significant transactions, such as those involving organization of environmental stimuli and stress resolution, conceivably inherit construct representation accordingly (cf. Neufeld, in press).

We turn for the moment to the assessment domain of psychophysiology. This domain is seemingly replete with measures poised for construct representation conferred by stochastic cognitive-performance models (cf. Tomarkin, 1995). Stochastic models of cognition and perception are capable of illuminating functions that bear, for example, on measures of evoked potential (Schweikert, 1989). Garnering construct representation for physiological measures, in terms of cognitive accompaniments, rests on a rigorous charting of cognitive transactions likely taking place during measurement epochs.

A case in point involves recent developments in high-field magnetic-resonance imaging (MRI). Patterns of brain activation across selected loci, and changes in activation during cognitive-task trials, can be recorded with remarkably high temporal and spatial resolution (Menon, Ogawa, Strupp, Anderson, & Ugurbil, 1995). However, the precision in specifying cognitive functions transpiring during MRI measurement periods (the "*functional*" side of *f*MRI) has lagged behind (Carter & Neufeld, 1998). Prospects of ultimately mapping one-to-one relations between functions and activation patterns, not to mention clinical abnormalities in these relations (cf. Cacioppo & Tassinary, 1990), have suffered accordingly. A factor complicating progress toward this goal has been the proclivity toward nonquantitative models of MRI-monitored cognitive events. The following representative example depicts this tendency, and its consequences.

An experimental paradigm introduced into fMRI research settings comprises conventional visual-search procedures. During each task trial, the participant scans a visual array of items (e.g., letters or digits) for the presence or absence of a prespecified target item. Within earlier trials of task performance, speed of detecting the presence of the target item (indicated, e.g., by a manual button press) is adversely affected by an increase in surrounding nontarget items. As trials progress, however, target-detection speed becomes unaffected by the number of nontarget

items that are present ("target pop-out effect"; see, e.g., Treisman & Paterson, 1984).

This eventual resistance of detection speed to an increase in surrounding nontarget items has been attributed to a change from serial to parallel inspection of items in the array. Tendered neurological substrata of the foregoing alteration have taken the form of modification with practice of neural circuitry recruited for task transaction (Ungerleider, 1996).

A more economical, and possibly valid interpretation of the performance shift, does not necessitate a qualitative transition in the structure of visual scanning (e.g., serial to parallel). Rather, items can be scanned in parallel throughout the range of practice. It is sufficient that the visual analysis of array items, dealt with in parallel, simply becomes more efficient. Technically, such an improvement in efficiency can involve a change of the processing system from one of "fixed-capacity with reallocation of resources" to one of "stochastic independence and unlimited capacity," or a "pure-death system" (described above; for relevant model mathematics see Townsend & Ashby, 1983, pp. 80–88). With the tenable prevalence throughout of a parallel processing architecture, hypothesized neurological substrata may be quite different from those hypothesized to underlie a switch from a serial to parallel structure. So too may be the corresponding interpretation of monitored fMRI patterns. Moreover, hypotheses embodying each interpretation of the change in task performance may be entertained; a critical asset of quantitative models is their highlighting of lingering ambiguities, and boundaries on existing knowledge, in this case regarding sources of switches in performance over trials.

With the astute application of pertinent quantitative tools, clinical cognitive psychologists are in position to supply key information about cognitive events paralleling measured neurological activation patterns. Included in this informational repertoire are (a) the architecture of candidate cognitive mechanisms (e.g., their serial vs. parallel structure) and (b) estimation of which component functions, such as stimulus encoding and scanning of memory-held information, are most prominent during constituent epochs of the measurement interval. Appropriate provision for the inevitable margin of error regarding such estimates is available by adopting models whose composition is duly stochastic.

The above example is taken from the rapidly developing "high-technology" field of fMRI. However, the essential points concerning formal models can be extrapolated to less exotic and expensive but nonetheless informative psychophysiological methodology (e.g., electromyographic measurement; Kukde & Neufeld, 1994). High-end technology in quantifying electrophysiological signals demands a similar level of quantification in the cognitive-perceptual domain of measurement.

On balance, prospects of process models of cognition contributing to construct representation of psychophysiological measures seem favorable. The following general strategy appears optimal; although it emanates primarily from our experience with high-field (Tesla-4.0) fMRI assessment methods, we believe that the approach may be of value beyond that specific setting (Carter & Neufeld, 1998; Neufeld & Williamson, 1996).

In the first instance, models representing task performance, such as speed or accuracy of visual search, above, are established among nonpatient samples. Such models then are perturbed so as to accommodate performance deviations among targeted patient samples. Minimal perturbation is sought, for parsimony. It typically appears unnecessary, for example, to modify the modeled structure of processing say from parallel to serial. Rather, alterations in selected parameters of a common structure appear sufficient. The procedure is roughly analogous to chemical titration, whereby the composition of an unknown substance is discerned according to the minimum amount of a standard substance required for a given reaction. In the present instance, the performance model is altered until the deviant performance pattern is reasonably matched. With this operational framework in hand, necessary alterations are taken to implicate cognitive functions changing with psychopathology, whereas model features remaining intact suggest functions apparently spared.

The task paradigm, and associated performance models, may then be productively imported into the setting of physiological recording. Aberrations in activation patterns among patients, corresponding with preestablished deviations in performance models from those of nonpatients, stand to imbue the former with significance regarding altered processing functions. As stated, changes in performance models ostensibly imply alterations in corresponding functions, such as retardation in encoding the physical or other properties of visual stimuli. Although subtle, and in the company of other contributing variables to be sure, the disorder-related changes in function may be symptomatologically or diagnostically significant (e.g., Neufeld et al., 1993). So too, therefore, may be associated changes in physiological activation. In this way, performance-model deviations may broker symptomatological and diagnostic significance to abnormal activation profiles.

Aptitude-treatment-intervention. A treatment for a given presenting problem produces a better response in a client with a given set of characteristics than in another with a different set of characteristics. A second treatment instead generates the superior response from the second client. This response scenario expresses simply the familiar clinical concept of aptitude-treatment interaction (ATI). It has been assumed that "aptitudes for competing treatments" comprise measurable individual differences.

For instance, some treatments for reducing stress of minor medical procedures may highlight information regarding details of the procedure and associated sensations, whereas others may downplay these details. Clients adept at processing such information, and translating it into pain-reducing maneuvers, may profit more from the treatment protocol dispensing procedural details; those less able, or disinclined for other reasons to process the available information, may be further ahead with the distraction-facilitating methods.

In a similar vein, individuals given to processing verbal information efficiently may progress more rapidly with therapy emphasizing verbal transactions, the opposite being the case for individuals more amenable to nonverbal, or imaginal transactions (Vallis & Bucher, 1986). Value of the alternate approaches, in other words, is suspected to differ among individuals according to the match between makeup of the procedure and traits of the individual.

Research on effects of matching treatment protocols to putatively relevant client characteristics has produced mixed findings (Dance & Neufeld, 1988; Ludwick-Rosenthal & Neufeld, 1988; Shoham-Salomon, 1991). For example, Ludwick-Rosenthal (Ludwick-Rosenthal & Neufeld, 1993) divided cardiac patients according to psychometrically measured preference for information surrounding cardiac catheterization (an invasive diagnostic procedure during which the individual remains conscious). The group preferring information responded relatively better to a preparatory treatment detailing the nuances of catheterization in advance, and less so to a preliminary treatment de-emphasizing procedural information. The Groups × Treatment interaction was statistically significant for selected measures of stress and coping; in that sense, results supported possible further work on the advisability of matching preparatory treatment to subject characteristics. The variance in response measures accounted for by the Groups × Treatment interaction, however, was relatively small, and could not be considered clinically substantial.

The ATI perspective on treatment selection may profit from process-model considerations. For an exemplary case, we return to the earlier instance involving process aspects of treatment effects. Alternate treatments may differentially affect process-model features across individuals. For some individuals, the impact of a given intervention may occur primarily with respect to one model feature or features (e.g., increasing v vs. decreasing k of a gamma-distributed remission process). For other individuals, changes in a different model features may predominate.[4]

Effects of a competing treatment alternative again may be expressed in terms of model features. In this instance, however, the primary changes in model features previously applicable to the first set of individuals may now apply mainly to the second set, and the opposite. The indicated treatment, of course, is the one whose main thrust takes place on the model features to be targeted.

Within this framework, a feature becomes targeted presumably according to its estimated relative value for the present client. For instance, given a gamma-distributed remission process, a comparatively low estimated v for an individual would suggest application of the appropriate v-enhancing treatment (see above). In the foregoing development, a certain emphasis has been placed on psychometric prediction of individual differences in model features susceptible to competing treatments. Like emphasis has been placed on estimating initial values of the germane model features ("treatment-targeted features") for individuals. Again, such estimates conceivably are abetted by feature-predictive psychometric measures. In all, then, psychometric measures putatively can help identify model features that need changing for given individuals and can help designate the treatment best suited to the individuals for effecting the indicated change.

Finally, the scores yielded by psychometric measures themselves may be subjected to process considerations. Specifically, item-response theory may be invoked (see Santor & Ramsay, 1998, this section). Rigorously defined expected values of process-relevant traits (θ in item-response theory terminology) are made available by these measurement models. Similarly, the most likely values of θ for an individual, given the person's configuration of responses to test-item options (Santor & Ramsay, 1998, this issue; Steinberg & Thissen, 1996) in principle can augment standard scoring methods for the process-related psychometric measures.

Challenges to Implementing Process Models in Assessment Settings

Potential benefits to using process models in assessment settings are considerable. At the same time, barriers to successful implementation are to say the least not trivial. Solutions established in pure research settings, such as those of cognitive science, can be instructive. However, their extrapolation to applied settings typically is not always a matter of course.

In this section, we briefly consider data characteristics that may deter process-model application. Avenues of potential resolution are outlined. Estimation of model features, and testing the verisimilitude of tendered models, are discussed. And aspects of the foregoing model fit and test strategies of special relevance to applied settings are considered.

An ubiquitous hazard to the valid application of process models comprises the presence of noise in the addressed data. Sources of variance in data relegated to the category, noise, entail influences extraneous to those that shoulder theoretical significance (see, e.g., Braithwaite, 1968). In other words, the designation of noise variance will vary with the research questions at hand. Noise sources by and large tend to cloud the operation of process models, potentially perturbing the identification of model features and evaluation of model validity. Note that where time-related variables are discrete, as in cases of transitional states (e.g., Batchelder, 1998, this issue), therapeutic sessions, or successive treatment intakes (n, as referred to above), issues of noise variance may be less salient than where continuous time t is involved. Certainly this statement characterizes our current experience in applied work on stress and psychopathology (Benn & Neufeld, 1996; Carter & Neufeld, 1998). The present emphasis, therefore, is on process models incorporating continuous time. Instances include those of cognitive assessment where the accent is placed on latencies of cognitive transactions, and where construct representation may be lent to psychometric and psychophysiological measures. For these reasons, a cognitive-assessment context emphasizing response latency conveniently is assumed in what follows.

As mentioned, the designation of noise sources of variance depends on the research question and setting. For example, individual differences represent a nuisance element for investigators focusing on nomothetic laws (see also, Batchelder, 1998, this issue; McFall & Townsend, 1998, this issue; Shavelson & Webb, 1991). Assessment psychologists, on the other hand, assign the opposite status to individual-difference variables. Students of nomothetic laws often circumvent the problem of participant-

[4] The effectiveness of a given intervention may also vary within individuals. For example, people who are recovering from addictions appear to cycle through a number of phases, with different interventions having greater effectiveness at different points (Prachaska, DiClemente, & Norcrosse, 1992). By analogy, in the present context, reference may be made to a concatenation of several gamma-distributed processes, with parametric variations on each one.

to-participant variants in the application of process models by obtaining extensive data from one individual at a time. A premium is placed on common aspects of model operation across individuals, possibly augmented with a summary statistic on the collective adequacy of model fit (e.g., overall χ^2 totaled across individuals with summed degrees of freedom; see also Delucchi, 1993; García Pérez, 1994). Although person-to-person variation in model expression is far from ignored in the nomothetic research setting, it may not be particularly relished, as it might be in the assessment psychology setting.

In applied settings, however, practical constraints often inhibit the gathering of extensive data from individual participants. Individuals may be too distressed for protracted participation, they may be unavailable for return sessions, or questions of theoretical or practical interest may require participation under stressing conditions. Therefore, amalgamation of data obtained over multiple sessions administered to individual participants may be forfeited in favor of amalgamation of data from multiple participants engaging in as little as a single session (cf. Suck & Holling, 1997).

Why the necessity for extensive data collection in the company of considerable noise variance? Variance in data detracting from that prescribed by an operative model, although detracting from the model's predictions, fortunately tends to attenuate with multiple observations. Sources of noise variance by and large are uncorrelated with model predictions, and therefore cancel, or compensate, across repeated observations (for a discussion of uses and possible abuses of data aggregation, see, e.g., Neufeld & Gardner, 1990).

It may be helpful to inspect the following statement of sources of variance in observed data:

$$\sigma^2_{observed} = \sigma^2_{process\text{-}model} + \sigma^2_{individual\ differences} + \sigma^2_{base\ process} + \sigma^2_{within\text{-}subjects\ residual}.$$

In this expression, the respective sources of variance are assumed to be independent or uncorrelated. The term $\sigma^2_{process\text{-}model}$ represents the variance prescribed by the stochastic aspect of the adopted process model. This term is theoretically meaningful to the degree that it incorporates substantively significant parameters (a point elaborated on in the Special Section Introduction article). For example, if a gamma-distributed item-encoding process were being examined (e.g., that of encoding visual-pattern features), this variance term would be specifically, k/υ^2, where k refers to the stages of encoding, possibly corresponding to k-item attributes (e.g., pattern features), and υ designates the rate of stage transaction. The term $\sigma^2_{individual\ differences}$ refers to individual differences in performance levels, or more precisely vis-à-vis the present context, to participant sources of differential elevation in latency of responding (e.g., differential reticence or propensity toward response registration). It should be underscored that such sources are deemed to be extraneous to systematic individual differences in process-model expression per se (detailed below). The term $\sigma^2_{base\ processes}$ indicates variance attributable to processes involved in registering the product of cognitive undertakings, and typically entails mainly rudimentary movement time for response registration, and possibly other response mechanisms. Such agents expeditiously are regarded as contributing to intertrial variance to the same degree for each participant. The fourth term $\sigma^2_{within\text{-}subject\ residual}$ on the right-hand side of the equation denotes within-subject latency variance emanating from sources apart from those prescribed by the stochastic model or base processes. These sources genuinely may deserve the label, "residual measurement error."

Observed quantities, including variances and means as well as distribution properties such as density and cumulative-distribution functions (described above and in the Special Section Introduction), therefore stand to be affected by sources of variance over and above those formally addressed by the theoretical process model. Sources associated with the second and fourth terms on the right-hand side of the above equation in particular fall into this category. Consequently, estimation of model features, and tests of model predictions to observed data are apt to fair poorly in the absence of provision for these extraneous, but often active sources of observed-data variance. Aggregation of performance data, by whatever means the exigencies of the research setting allow, takes on importance because of the involvement of these extra-model sources of variance. Provision for random individual differences in practice appears especially weighty. At the same time, note that within-subject residual sources of variance are attenuated in the computation of individual participants' means, but not necessarily their variances computed across trials, nor distribution properties. In practice, lingering effects of residual within-subject variance may influence estimates of the latter two features, but not sufficiently to dislodge the verisimilitude of otherwise viable models.

Additional concerns come into play, however, when aggregating data across individuals: there may be systematic differences in model features within the aggregated group (see also, Luce, 1997). Amalgamating data from systematically differing enclaves of participants risks generating an amalgam of model features that represents none of the contributing enclaves (e.g., Neufeld & Gardner, 1990). This dilemma is an instance of data collectives embodying probability mixtures, or components of performance data. The difficulty will occur if subgroups of individuals differ with respect to model structure (e.g., parallel vs. serial architecture of processing), or, if they differ with respect to values of parameters embedded in a shared structure. For example, performance of some individuals may be expressed in terms of gamma-distributed processing latencies, and that of others may be expressed in terms of a parallel-processing pure-death system (above). Alternatively, the performance latencies of each and every participant may approximate a gamma distribution; the value of one or both of its parameters, however, may systematically vary across the participants. Estimating parameter values for each participant (or for a subsample of participants) with a view to identifying trends, even though measurement error precludes adequate model fit at the individual level, is one way to address this issue.

Observe that the possibility of systematic components, or probability mixtures of model features, is not unique to aggregation across participants. Although not always addressed, data from individual participants, obtained over multiple experimental sessions in the pure research setting, are susceptible to the

operation of systematic shifts in processing strategies. In other words, components now attend sessions, within subjects, rather than subjects, within sessions (cf. Burbeck & Luce, 1982).

From the perspective of psychometric measures, data protocols from individuals, or sessions, should represent *parallel*, rather than *heterogeneous* measures of process-model distributions. Prior to aggregating data from a set of individuals, some estimate of homogeneity is desirable. In the case of moments, for example, a nonsignificant Kolgomorov–Smirnov test for departure from a single distribution of participants' means or across-trial standard deviations may be a minimal preaggregation requirement. Where multiple distribution-values are involved, as in the case where a moment-value is computed for each of several conditions of performance, or where multiple time intervals are used in calculating cumulative-distribution-function values [$F(t)$, above], checks on homogeneity of response profiles may be performed (e.g., certain uses of coefficient alpha, e.g., Neufeld & McCarty, 1994; see Schmitt, 1996; also, profile analysis or components analysis, Benn & Neufeld, 1996, 1997). Aggregation taking place across similarly performing participants in principle permits inferences to classes of individuals, thus identified. Returning to considerations of construct representation of psychometric and of other measures, the relevant associations appropriately involve process-model features educed for the respective enclaves; these features, then, potentially are implicated for a given client with the enclave-related psychometric scores.

It is possible to estimate the involvement of differential model expression in a data collective by designating certain model features as random, rather than fixed. Considerable flexibility can be gained by allowing various aspects of a model to vary. Apparently complex functions may entail two simple functions, or one simple function with varying parameter values. Specifically, estimation of feature-related individual differences in principle can be made within a stochastic-process motif, through the use of probability mixture models. For example, differing model structures (e.g., parallel vs. serial) may be active by differing amounts within the participant sample (Neufeld, 1994; cf. Yantis, Meyer, & Smith, 1991). The probability that processing assumes one structure versus another may be assigned a parameter, say π, the probability of the opposite structure necessarily being $1 - \pi$. The amount of heterogeneity then is indicated by the estimated value of π. In a similar vein, the rate parameter v say of a gamma distribution, itself may be gamma distributed, with parameters r and u (the latter once again being a continuous analogue of the parameter k; Neufeld, 1991). In this case, the mean value of v is u/r. The variance in v may be substantial or trivial depending on its value as estimated according to u/r^2. In like fashion, the parameter p of the Pascal distribution may be expressed as the random variable $\exp(-z)$, where z is gamma distributed, varying from 0 to ∞. Given a gamma-distributed z, with parameters r and u, the mean value of p is $r^u/(r + 1)^u$, and its variance is $r^u/(r + 2)^u - [r^u(r + 1)^u]^2$. Moreover, hierarchical mixtures are available, where multiple parameters (e.g., v as well as k of the gamma distribution) take on random status. Further details on probability mixtures in applied process modeling, including some Bayesian considerations, are available in other sources (Batchelder, 1998, this section; Neufeld, 1994, 1996). Note that methods of estimating parameters of mixture models by and large are extensions of those used for fixed-parameter distributions, below (see, e.g., Batchelder, 1998, this issue).

An important strategy in addressing model-pertinent features of empirical data is to use multiple methods of estimation, where possible. Consider for example, computation of certain distribution properties from observed values. Calculation of the distribution function $F(t)$, for one, may be carried out using complementing methods. It is possible to simply tabulate the observed proportion of events occurring at or before t. Another method consists of fitting a density function $f(t)$ to empirical observations (e.g., Silverman, 1986). The recovered function may then be integrated (numerically or analytically) for selected values of t, resulting in additional estimates of $F(t)$. A further method hopefully yielding convergent results entails the computation of the hazard function $H(t)$, which is $f(t)/[1 - F(t)]$ (Bloxom, 1984; see Special Section Introduction for elaboration). This function then is inserted into the following formula for $F(t)$:

$$1 - \exp\left[-\int_0^t H(t')\,dt'\right].$$

Similar considerations apply to estimating parameter values of parametric process models, such as that specifying the gamma distribution. Again convergent evidence for tendered values involves complementing methods of estimation. The parameters of the gamma distribution, for instance, can be estimated by submitting the obtained data to "moment-matching" and "maximum-likelihood procedures" (Evans et al., 1993). Computerized parameter-search algorithms also can be used. In this case, various versions of correspondence between model predictions and observed data are expressed by available functions (technically, cost functions) to which the algorithms are administered (e.g., Neufeld, 1996). Regardless of the cost function invoked, parameter values yielding maximum correspondence are sought. Again, convergence of resulting values is the issue at hand. For additional observations on model fitting and testing procedures, see the other contributions to this Special Section.[5]

Mention was made, above, of the possible utility of probability mixture models in dealing with systematic individual variations in model expression; in this case, parameters of process-model distributions themselves have randomly distributed values. Once more, multiple avenues of model testing and parameter identification are in order (cf. Bamber & van Santen, in press; Cutting, in press). The possible random status of process-model parameters may be judged in part by using information from alternate data treatments. The point once again can be illustrated with reference to the gamma distribution, but generalizes elsewhere. The hazard function $H(t)$ of the gamma distribution will be nonmonotonic with increasing t if values of one or both of its parameters are randomly distributed [contra the usual monotonic increase in $H(t)$ of the gamma distribution; for a

[5] Extensive treatment of model testing is dealt with in a special issue, "Methods of Model Selection," of the *Journal of Mathematical Psychology* (in press).

plot of the latter function, see the Special Section Introduction]. Variance of the distribution's latency values, however, is predicted to change in qualitatively distinct ways across specified conditions of measurement (e.g., cognitive-task demands designed to elevate processing stages), depending on whether v is fixed or random, but not depending on whether k is fixed or random (Neufeld & Williamson, 1996; Vollick, 1994).

Concluding Comments

Available and continuously emerging process-model methodology makes for a potentially penetrating arsenal of analytical tools for addressing the dynamic character of clinical transactions. As is the case with any analytical strategy, however, the fidelity of informational returns is in lock step with the rigor of application. Additional considerations come into play when formulating assessment problems in terms of stochastic process models. When all is said and done, like with other analytical tacks, immediate practical value is judged by the usual criteria, such as predictive utility, and incremental predictive utility over existing procedures.

Much has been said about construct representation in the above developments. In the applied arena, however, considerable importance is attached to "nomothetic span" (see Embretson, 1983). In the present context, nomothetic span refers in good part to the strength of association between model predictions and observed data, and consistency in predictive accuracy over occasions and contexts of assessment. In the clinical setting, observed data routinely comprises that of individual clients. Model testing and parameter-value identification may require aggregation of data across participants, as described above. Where the unit of prediction is the individual, however, model utility is judged against disaggregated data (Lees & Neufeld, 1994). If a remission process is addressed, for example, acceptable correspondence between modeled distribution properties and moments, and observed counterparts emanating from individual data points, may await model fine-tuning (e.g., modifications to model structure and values of distribution features). The same result may hold in the case of cognitive-assessment models, where the protocol of response latencies for individuals comprise the focal predicted data (e.g., Carter & Neufeld, 1998). On the other hand, when the desired level of inference is to aggregate data, such as that from a relatively homogeneous group of patients, or an institution whose data collective is amenable to a selected model structure and parameter values, predictive efficiency may be substantial at the outset (cf. Luke & Homan, 1998, this issue).

A vital service rendered by quantitative process and other models is the highlighting of continuing perplexities in need of attention. Lingering ambiguities and boundaries on existing knowledge are laid somewhat bare. Assumptions are exposed by the formal, axiomatic reasoning imposed. The distillate of a modeling enterprise may consist of a set of tenable models, none of which is decisively rejected by observation; certain process architectures can be empirically equivalent to fundamentally different architectures (see, e.g., Townsend & Ashby, 1983, chapter 14). Features held in common by the reduced set of retained models nevertheless may be informative. For example, although several models of cognitive performance may accommodate deviations evinced by psychopathology, anomaly in a similar type of parameter, or other model aspect, may emerge in each instance (e.g., one depicting processing stages vs. one depicting "channel-capacity," or rate of stage completion).

Continual progress is being made in deciphering theoretically important and clinically relevant model composition. With sustained involvement, and because quantitative developments usually transcend content areas, clinical measurement technology not only should reap the benefits of ongoing advances, but it stands to become an active contributor to the armamentarium of model diagnostics and effective application.

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