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A Shock Model for the Pregnancy at Pre Partum and Post Partum

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Abstract
In this paper, a shock model for the effect of stress in terms of cortisol is studied. Assume that shocks arrive according to a Poisson process. Shocks are events which cause perturbation to the system, leading to its deterioration and consequent failure. The Cartisol Awakening Responds (CAR) at pre partum and post partum are compared by this model.

Keywords: Cartisol Awakening Responds (CAR), Delivery, Hypothalamic-pituitary-adrenal (HPA), Pregnancy, Shock model.

I. Introduction

Among the many approaches to modeling deteriorating systems, shock models have found favour with reliability analysis because of their tractability and wide applicability to diverse areas [3,5,12].

1.1. Notations

Z:	Random variable denoting the time between two successive shocks.
$F(.),\overline{F}(.)$:	Cumulative distribution & survivor function of Z.
W:	Random variable denoting time between two successive failures.
$k(t), K(t), \overline{K}(t)$:	Probability density, cumulative distribution and survivor functions of W.
D:	Random variable denoting the threshold value.
G(.):	Cumulative distribution of D.
$N(t)$:	Counting variable denoting the number at failures in (0,t)

1.2. Assumptions of the Shock Model

A new system is put on operation at time $t = 0.$ The system on failure is repaired and successive repairs are assumed to take negligible amount of time.

The system is subject to shocks. The interval between shocks % are assumed to be independently and identically distributed with distribution function F(.).

A shock is classified as a nonlethal shock if the time elapsed from the previous shock to this shock is greater than the threshold *D*. A shock is lethal if it occurs within *D*. A lethal shock results in system failure leading to its repair.

Threshold time *D* is a random variable with distribution function G(.).

The shock arrival times and the threshold time are independent of each other.

1.3. Characteristics of the Model

We first obtain the probability density function of W at the time between two successive failures. From the stated assumptions, the survivor function $\overline{K}(t)$ of the time between failures satisfies the integral equation

$$\overline{K}(t) = \overline{F}(t) + \int_0^{\tau} f(\tau)G(\tau)\overline{K}(t-\tau)d\tau \quad \text{... (1)}$$

Equation (1) may be derived as follows. The event $\{W > t\}$ can be decomposed into two mutually exclusive events as given below.

(1) The first shock itself occurs only after *t*, the probability of which is $\overline{F}(t).$

(2) The other possible event is a conjunction of the following three events.

(a) The first shock occurs at some instant $\tau \in (0,t]$ the corresponding density being $f(\tau)$

(b) The threshold time starting from $t = 0$ is over by time $\tau,$ the probability of which is $G(\tau)$ and

(c) In the remaining interval $(\tau,t]$ of length $(t-\tau)$ there is no failure, the probability of which is $K(t-\tau).$ Integrating over all

possible $\tau \in (0,t]$ we obtain the second term of equation (1). Simple differentiation of (1) yields the probability density $k(t)$ of the random variable W as

$$k(t) = f(t)\overline{G}(t) + \int_0^t f(\tau)G(\tau)K(t-\tau)dt$$

When the system is subjected to the same kind of shock each time, the threshold time of the system is likely to remain a constant, a case discussed by Yeh Lam [5, 13]. Under such a scenario, we consider a few models for different shock arrival distributions.

First, we assume the shock arrivals are according to an exponential density with mean $\frac{1}{\lambda}$. The relevant statistical characteristics can be derived as

$$K(t) = \lambda e^{-\lambda t} \sum_{N=0}^{\infty} \frac{\lambda^n}{n!} \{(t-nd)^n U_{nd} - (t-(n+1)d)^n U_{(n+1)d}\}$$

$$E(W) = \frac{1}{\lambda(1-e^{-\lambda d})}$$

$$Var(W) = \frac{2(1-e^{-\lambda d}) + 2\lambda e^{-\lambda d}(\lambda d e^{-\lambda d} - e^{-\lambda d})}{\lambda^2 (1-e^{-\lambda d})^2}$$

$$M(t) = E(N(t)) = \lambda t - \lambda e^{-\lambda d}(t-d) \qquad t > d$$

$$Var(N(t)) = \lambda t(1-e^{-\lambda d}) + \lambda d e^{-\lambda d} - (\lambda d e^{-\lambda d})^2$$

II. Application

Hypothalamic-pituitary-adrenal (HPA) responses to physical or psychological stress are reduced during the postpartum period [10, 14]. This phenomenon may help the mother to conserve energy required for lactation, protect against stress-associated inhibition of lactation, relieve psychological stress, and enhance her immune function [1,6]. However, alterations of HPA activity have also been associated with mood disturbances and several puerperal disorders, including postpartum blues and postpartum depression [2]. The factors that may cause these HPA changes during the postpartum period are largely unknown. To identify them, most human studies focused on maternal characteristics postpartum [14].

The maternal organism undergoes remarkable neuroendocrine changes during pregnancy, optimizing fetal growth and development, protecting the fetus from adverse exposures, and preparing the mother for timely parturition. More specifically, gestation dramatically affects the maternal HPA axis, leading to increased basal levels of corticotrophin-releasing hormone (CRH), adrenocorticotropin (ACTH), bound cortisol, unbound cortisol in human plasma [7, 4], and a more pronounced salivary cortisol awakening response. The physiological consequences of this increase in cortisol remain a matter of debate, but most discussions have focused on effects on the fetus [8]. However, another physiological effect of increased cortisol concentrations at the end of pregnancy may be related to postpartum HPA reactivity. The CAR represents the steep increase of cortisol secretion within the first 30 min after awakening, usually leading to the highest cortisol concentrations throughout the day [9, 15]. As basal cortisol levels are already markedly increased during late pregnancy, the cortisol increase after awakening leads to an even stronger exposure to cortisol during the prepartum period. This exposure may profoundly change the regulation of the maternal HPA axis, with lasting effects persisting throughout the postpartum period.

Our primary objectives were to determine, (i) whether the CAR during late pregnancy, as an indicator of the markedly increased cortisol concentrations at the end of pregnancy, predicts maternal HPA responsiveness to a psychosocial stress test postpartum, (ii) whether this prediction is specific to the CAR during pregnancy or a feature of the CAR independent of pregnancy.

III. Methodology

We conducted a longitudinal study with an experimental component. Pregnant women were recruited by local advertisements between the 20th and the 36th week of gestation (WG) for paid participation in a study of lactation and stress. Twenty-two healthy women participated in this study (mean age (standard deviation, SD): 30.1 (4.2) years), including 10 (45%) primiparous and 12 (55%) multiparous women, which is comparable to the general population of women giving birth. Participants underwent a medical examination and a diagnostic interview before entering the study and were considered eligible if they were free of chronic diseases, mental disorders, medication, smoking, and drug or alcohol abuse. None of the women had resumed menses before the day on which the assessment at 8 weeks postpartum was completed. The assessments took place at three time points during the peripartum period. In the 36th WG and at 6 weeks postpartum, women collected saliva and responded to questionnaires at home. At 8 weeks postpartum, women reported to the laboratory to respond to further questionnaires and were exposed to standardized psychosocial laboratory stress protocol.

The salivary CAR was assessed in the 36th WG and at 6 weeks postpartum. We instructed participants to collect saliva samples at home 0, 30, 45 and 60 min after awakening (first timed wake-up sample at 6.30 am, with use of an alarm clock if necessary, as it has been shown previously that

spontaneous awakening versus alarm awakening does not affect the CAR [11, 16]. We asked the participants to choose a weekday to collect samples and to abstain from consuming any food or brushing their teeth prior to the four samples in order to prevent any contamination. Adherence to protocol was assessed by self-report questionnaire.

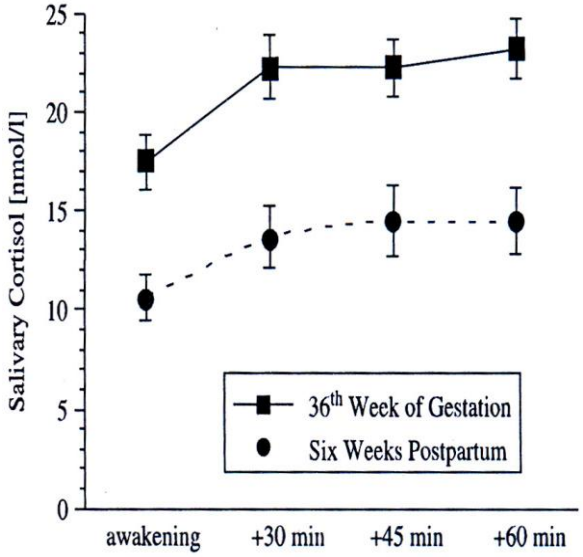

Figure 1. Cortisol awakening responses. Plot of the salivary CAR (mean ± standard error of mean, SEM) at 36th WG, week of gestation (solid line, squares; n=22) and at 6 weeks postpartum (dotted line, bullets; n=15). *Statistically significant increase in salivary cortisol from awakening to 30 min after awakening (paired one-tailed t-tests; 26th WG: t(21) = 3.48, P – 0.001; 6 weeks postpartum: t(14) = 2.05, P = 0.029).

IV. Mathematical Result

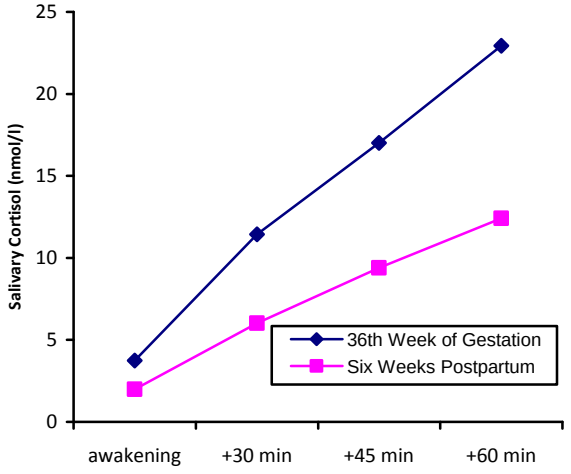

Figure 2. Salivary Cortisol (nmol/l) Vs Time. By using mathematical model

V. Conclusion

Shocks are events which cause perturbation to the system, leading to its deterioration and consequent failure. From the shock model this study found that there is an association of the CARS between pre partum and post partum. Also, this study found that a feature of CAR is dependent on pregnancy as the upper curve increases more than the lower curve when the time *t* increases.

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