Catastrophe Theory and the Human Sexual Response

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Abstract

A nonlinear differential equation model and its associated catastrophe is shown to model the simplest version of the sexual response of humans. The mathematical model is derived via well-known and non-controversial aspects of sexual orgasm as can be found in the literature. Two different and independent derivations of the equation are given; the equilibrium-oscillation model and the slow-fast dual equation model.

Keywords sexual response, nonlinear differential equation, catastrophe

1.Introduction

Not every process in the world is continuous and slowly changing. The particular combination of continuous (and often slow) changes vs discontinuous (rapid) changes (effects) can probably be best described/modeled in terms of a new theory invented by Thom[1975] and which has benefited from the many contributions by E.C. Zeeman [1977]. This branch of mathematics is known, appropriately enough, as Catastrophe Theory. The literature is replete with scientific papers applying catastrophe theory to various social, psychological and biological phenomena [for example, Zeeman,1977].

Many more books have been written on nonlinearity and complexity in recent years [see for example Hubey,1999]. Catastrophe theory is in some ways the ideal tool to analyze phenomena with rapid or discontinuous changes, and has been used for various biological models by Thom [1975]. In mathematical modeling of the sexual response of human beings the phrase "sexual tension" is used for physical-correlates of the term which can be summarized as the occurrence of relatively intense physiological arousal, enumerated usually as consisting of, extensive changes in cardiovascular, respiratory, vasocongestive, muscular, and other physiological activities. Some kind of fuzzy function of these variables can thus be used as a proxy.

2. Dual-Enervation Model

Fig. I is a reconstruction [from Masters & Johnson,1966] of the male sexual response (the simpler of the two). The corresponding one for the female is more complicated and will probably have to be constructed as a combination of the solutions for the simpler case. A cusp catastrophe, Fig. II, has essentially the same information as in Fig. I. Time is implicit. The phase point moves along the (response) surface.

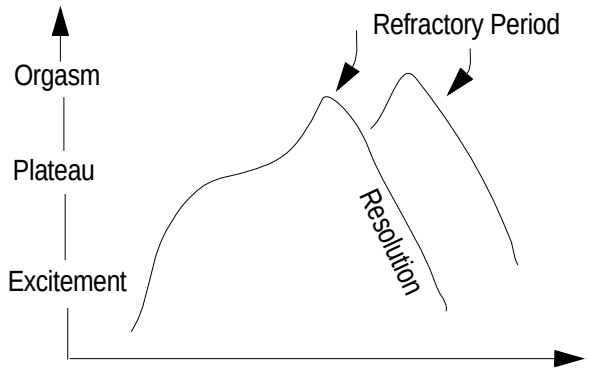

Figure I: A suggestive diagram reproduced from Masters and Johnson[1969].

As for experimental evidence, and for thoughts on the subject by other researchers, we note that Kaplan [1979], for example, considers the emission phase to be controlled entirely by sympathetic innervation in particular, alpha-adrenergic receptor stimulation of the smooth muscles in the male accessory glands. According to Kaplan, the emission response is experienced subjectively as "ejaculatory inevitability," but is not intrinsically pleasurable. Ejaculation, on the other hand, is controlled by somatic innervation of striated muscle groups at the

base of the penis, find is accompanied by "the typical pleasurable orgastic sensations" [1979: 20]. Kaplan also postulates the existence of an "orgasm center" in the sacral spinal cord, which is said to coordinate both phases of the response. As for the mechanism of trigger, researchers have specified myotonic or vascular responses as orgasm triggers.

Sherfey's proposal [1966] is that orgasm is a spinal reflex that is triggered by firing of the stretch receptors in the pelvic musculature, while Mould[1980] suggests that a principal effect of vasocongestion is to cause biasing of the gamma fusimotor muscle spindles. Once the muscle spindles become highly biased, and a dynamic stretch reflex is initiated in the alpha fusimotor. The chemical basis specifically has been investigated by Brindley [1983] who has shown that alpha-adrenoceptor blockers produce reflex erections. It has also been shown that the injection of papaverine also causes erections by [Virag & Virag 1983, Zorgniotti & Lefleur 1985].

The empirical evidence suggests that we should try the simplest catastrophe and that we should try to account for the highest level phenemena since the lack of precise and accurate measurements that are necessary for creating mathematical models do not exist. The cusp catastrophe is shown on Fig. II.

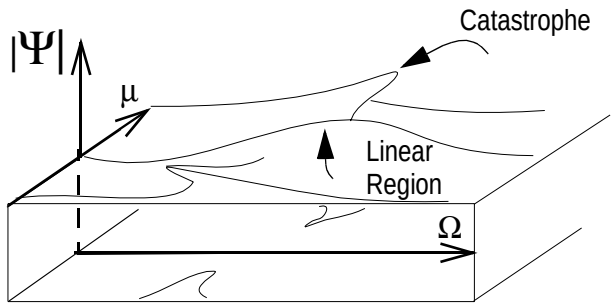

Figure II: Catastrophe Surface
The resonance occurs at $\Omega = \omega$.

The variable Ψ represents 'sexual tension', Ω is the frequency of the 'excitation', and μ is some nonlinearity parameter. It is seen in Fig.(III) that the response as a function of the driving frequency Ω is not single-valued. In Fig. (II) we see the response surface for $\mu < 0$, $\mu > 0$, and $\mu = 0$. The last case corresponds to the linear model Fig. III is a cross section from Fig. II it clearly shows the *jump phenomenon* a well-known from physical (mechanical and electrical) systems. The *jump* is a rapid change (relatively speaking) in the amplitude of the response. With the increasing frequency of excitation, there is a sudden change in some of the correlates of sexual tension (for example muscle tension) at the point of orgasm, and there follows a cycle of sudden increases and decreases of muscle tension which is normally associated with orgasm (along with the other gender specific responses such as emission.)

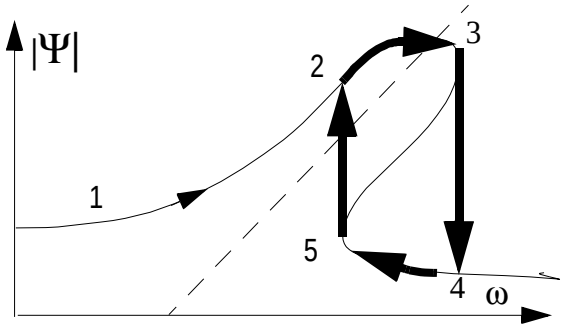

Figure III: The Catastrophe:
This is the cross-section of Fig. II.

The sudden (almost) discontinuous jump cycles 2-3-4-5-2-3-4-5... is what is modeled as the orgasmic phase, as shown in Fig. III. However, another path can be taken on this surface due to psychogenic changes, which, of course implies that a more realistic model should have time-dependent coefficients so that the response surface can change in time due to psychological effects.

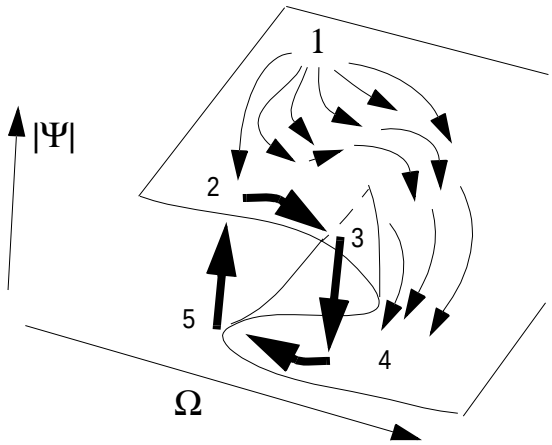

Figure IV: The Catastrophe Surface for the control parameter m Š 0. Rough direction for the frequency of excitatation W, and the magnitude of the of the response (|Ψ|) (sexual tension) is shown.

The slow dissipation of physiologic residuals of sexual tension can then be thought of as the change in the response surface (Fig. IV) or the motion of the phase/state point to a lower state of tension. Furthermore, in Fig. IV we can see that there are alternative paths from point 1 to point 4. The simple and idealized case solved here corresponds in a macroscopic/averaged sense to what is normally observed.

3. A Simple Oscillation Model

The basic model analytically can be thought of as something like the opponent processes of psychology which seems to reflect most of all the fact that there is negative feedback loop since all controllable systems must exhibit such a capability. Suppose that sexual excitement and orgasm is mediated by a two-factors (which are still unknown) as already conjectured by Davidson [1980], Weiss [1972], Rosen & Beck [1988] etc. We can start our modeling by assuming the simplest kind of a dynamic dependence, by which we mean a coupled set of ordinary differential equations using two variables, $\dot{V} = G(U, V)$ and $\dot{U} = F(U, V) + N(t)$. We assume that normally there is some kind of static or dynamic stasis (or equilibrium) in that we can write the variables as $u = U - U_0$ and $v = V - V_0$ where the U_0 and V_0 are the *equilibrium* values so that $F(U_0, V_0) = 0$ & $G(U_0, V_0) = 0$ .and thus

3) $\dot{u} = F(U_0 + u, V_0 + v) + N(t)$

4) $\dot{v} = G(U_0 + u, V_0 + v)$

Now, expanding the rhs in Taylor series we obtain

6) $\dot{u} = -m_1 u - m_2 v + N(t)$

7) $\dot{v} = -m_3 u - m_4 v$

with $m_1 = \frac{\partial}{\partial U} F(U_0, V_0)$, $m_2 = \frac{\partial}{\partial V} F(U_0, V_0)$,

$m_3 = \frac{\partial}{\partial U} G(U_0, V_0)$, and $m_4 = \frac{\partial}{\partial V} G(U_0, V_0)$

where we've assumed that the signs of the partials are negative because of the negative feedback effect that is necessary for stability of systems. We can simplify the equations by differentiating (6) to obtain

8) $\ddot{u} = m_1 \dot{u} + m_2 \dot{v} + \dot{N}(t)$

and substituting for $\dot{v}$ from (7) and solving for v in (6) and also substituting for v in which we obtain

9) $\ddot{u} + A\dot{u} + Bu = m_3 N(t) + \dot{N}(t)$

with $A = m_1 + m_3$ and $B = m_1 m_3 + m_2 m_4$ which is the damped harmonic oscillator. We note that this equation is still linear and that the forcing should be modeled via a sinusoid (which is the simplest approximation of a periodic/cyclic stimulation or excitation.) We could have just as easily defined N(t) as an integral of a sinusoidal function since the state of sexual excitation is a function of the past excitations or we could have made N(t) a convolution integral with some kind of weighting. Only the future experiments will tell which of these is the best model since about the only thing we know now is that it is essentially periodic. The spectral response of the linear harmonic oscillator, as is well known (and shown in Fig. V), has a high response at the natural frequency ω.

4. Slow-Fast Dual Mechanism

There exists yet another derivation for the model. That the lower life forms (at least the males) can have something like orgasm would indicate that the primitive part of the *triune brain*[McLean,1973] is involved and that this would implicate the limbic system. Furthermore that there can be emission without orgasm is indicative that there seems to exist two separate systems that work in tandem to produce orgasm and that another approach to the derivation of the mathematical model would be fruitful. We can take an approach similar to that pioneered by Zeeman [1977] in that we can posit the existence of a fast equation and a slow equation which work together to produce catastrophes. Following the ideas of Zeeman [1977] the model can be written as a system of two first degree equations

10) $\dot{u} = -\alpha v$

11) $\dot{v} = av + bu^3 + g\sin(\Omega t)$

The set above is equivalent to the single second degree differential equation given in eq.(1). We can differentiate eq. (10) and substitute for $\dot{v}$ (from 11) to obtain eq.(1) with $\omega^2 = ab$, $2\xi = \alpha a$, backhand A=ag. The constant a is large so that eq.(12a) is the *fast equation* [see Zeeman,1977]. The constants a,b, and g are small so that eq.(11) is the *slow equation*. Hence we have $\xi < 1$, ω can be large, and b is also small. The linear version (i.e. g=0) is equivalent to the set of equations (10-11) with $\xi = (m_1 + m_3)/2$, $\omega^2 = m_1 m_3 + m_2 m_4$, b= 0 and with the substitution of an oscillatory forcing or excitation $g\sin(\omega t) = m_3 N(t) + dN/dt$. It can be shown that the set of equations below

12) $$\dot{u} = 2\xi u + \alpha v$$

13) $$\dot{v} = bv + cu^3 + g\sin(\Omega t)$$

is also equivalent to eq.(1) with b, c and g given as above. The same reasoning as above shows that the first equation is again the fast equation and the second, the slow equation. What is important in these models is that as two one-dimensional equations they are both of the standard/classical *opponent theory* of psychobiological processes, of which the bipolar theory, and the somatogenic and psychogenic response type, as in Masters and Johnson or Kaplan or Bancroft are specific examples. We can, as a first approximation, assume that psychogenic processes can change more rapidly than somatogenic ones. In all cases, the models lead to the second order nonlinear DE given below:

1) $$\ddot{\Psi} + 2\omega_n \xi \dot{\Psi} + \omega_n^2 \Psi + \mu \Psi^3 = A\sin(\Omega t)$$

where the overdot indicates derivative with respect to time and which is better written as

2) $$L_0\Psi + \mu\Psi^3 = L_0\Psi + L_1(\Psi) = \sin(\Omega t)$$

where $L_0 = \frac{d^2}{dt^2} + 2\omega_n \xi \frac{d}{dt} + \omega_n^2$ is the linear damped harmonic oscillator operator, $\xi \ll 1$ and L_1 is the nonlinear operator. The source term in the differential equation above is the simplest kind of rhythmic (periodic) physical stimulation. The nonlinear differential equation above, known as the Duffing equation/oscillator named after the first person to have studied its properties, cannot be solved in closed form. Furthermore, we know that for the linear case (i.e $\mu = 0$ see Fig. V) there is no jump but there is a resonance phenomena, and since the response surface does not have the tilt but rather the common DHO response, there is no orgasm but rather a slow decrease of the amplitude with increasing driving frequency. Hence the nonlinearity coefficient, can represent a particular type of dysfunction in which there is failure to achieve orgasm despite sustained excitation.

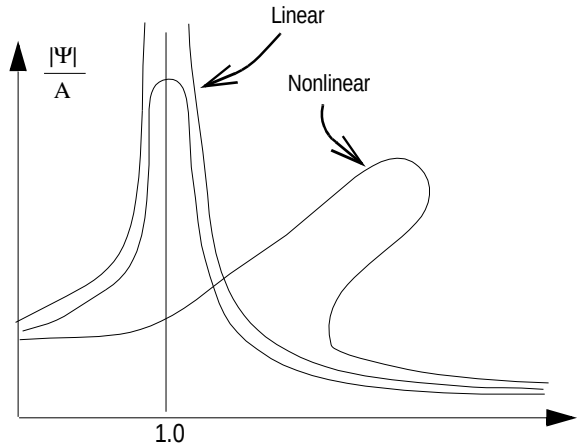

Figure V: Linear Response Patterns: The linear response corresponds to μ=0. The parameter ξ is the damping (which to a first approximation may be taken to be synonymous with inhibition which is not necessarily equivalent to one being of psychosomatic origin). The maximum amplitude at resonance (Ω/ω=1) is infinite for the undamped case (ξ=0). But for positive damping (ξ>0) the response curves to the left and right of the dotted line at Ω=ω meet.

On the other hand the damping coefficient is the determiner of the maximum tension that a person can tolerate before the point of no return is reached, and is therefore the parameter that one would associate with concepts such as inhibition, 'magnitude of orgasm', etc. The smaller the damping, the larger the magnitude of the peak point (point 3) so that typically this could be associated with the subjective feelings of satisfaction, and could also be measurable objectively via the rate of dissipation of the physiologic residuals such as vasocongestion. We could go further and posit whether the response

surface also changes as a result of orgasm to (almost completely flatten) or whether the response surface stays the same but the state point moves along the response surface to a lower level of tension.

5. Conclusion

It can be seen from the equations that the psychogenic (and some somatogenic) effects are being represented by the coefficients of the DE while sexual tension can be approximated as the magnitude of the amplitude of the response. The response surface slowly develops via psychogenic and somatogenic excitation, and that purely physiological excitation could trigger only a single component of a multiple complex system. Furthermore it would add more evidence to the view that this response surface is not really a always in existence as shown in the figure as a part of the human body system (as in mechanical or electrical systems) but that it only develops during the excitation phase so that the coefficients themselves are functions of the organism or are representative of various components. Hence we can see that, using time as a proxy (to simplify) for these as yet unmeasurable variables, we should make the coefficients functions of time which immeasurably complicates the equation.

The equation that is given as a model for sex and orgasm, as complex as it may seem, is only the simplest kind of a mathematical model of an immensely complex behavior. Of course, the fact that the catastrophes are a high level (i.e. simplified views) descriptions has already been noted [Zeeman,1977]. Naturally the biochemical aspect if a part of the whole phenomenon of sexual excitation and should be accounted for (eventually) by mathematical equations, but the ordinary differential equation model shown here and its associated geometry (catastrophe) is a high level description and cannot account for low level phenomena. When more is known, the relationships of the coefficients of equation (1) to the lower level biochemical, mechanical and electrical underpinnings may be established. As for the reported marked alterations in consciousness in the literature, explanation proposed by Davidson [1980], is again a two-factor model, which not surprisingly has to do with rapid fluctuations or oscillations in autonomic balance from parasympathetic (trophotropic) to sympathetic (ergotropic) dominance and that this is the cause of the major alteration in conscious experience. Drawing upon Gellhorn's [1957] original hypotheses on the effects of autonomic "imbalance" on central arousal states Davidson suggests that high levels of sexual arousal result in simultaneous (i.e., "nonreciprocal") activation of both autonomic systems, and a consequent change in central arousal. The word "imbalance" is obviously an attempt at description of changes in the equilibrium of a system or fluctuations or oscillations between two poles or two phases. Gellhorn's model would also predict a rapid "rebound" effect following strong ergotropic activation which may account for the state of quiescence usually associated with the refractory period.

Finally, Davidson notes that major changes in autonomic balance appear to be associated with a shift to right (nondominant) cerebral activation, which is consistent with the results obtained by Cohen et al 1976]. This corresponds to the unfolding of the response surface so that it is flat, and thus the disappearance of the orgasmic platform. The coefficient ξ is a control parameter which probably has something to do with the erotophobia-erotophilia variable of Byrne, since the response is greater for small values of ξ. Appropriately enough, this parameter in physical systems is the amount of "damping" in the system so that high values will dampen down the oscillations and hence the response even at resonance. For values of $\mu = 0$, the system is linear and an increase in the frequency of the excitation cannot cause a jump phenomena. Instead a continuous increase in amplitude will be seen until resonance and the response will decrease if the frequency is increased, so that there could be a slow decrease in tension but no orgasm. Therefore there is capability in the mathematical equations for modeling cognitive and psychological aspects of both normal response, and various dysfunctions.

5. References

Bancroft, J.H. *Human sexuality and its problems.* New York, Churchill-Livingstone, (1983)

Barlow, D.H. (1986) Causes of sexual dysfunction: The role of anxiety and cognitive interference. Journal of Consulting and Clinical Psychology, 54, 140-157.

Brindley, G.S. (1983) Cavernosal alpha-blockade: A new technique for investigating and treating erectile impotence, British Journal of Psychiatry, 143, 332-337.

Byrne, D. (1977) The Imagery of Sex, in J. Money & H. Musaph (Eds.) Handbook of sexology (pp.327-350) Amsterdam, Elsevier.

Byrne, D. (1983) The antecedents, correlates, and consequents of erotophobia-erotophilia, in C. Davis (Ed.) Challenges in sexual science (pp.53-75) Lake Mills, IA, Graphic.

Byrne, D. (1986) The study of sexual behavior as a multidisciplinary venture, in D. Byrne & H. Kelly (Eds) Alternative approaches to the study of sexual behavior (pp. 1-12) Hillsdale, NJ, Erlbaum.

Byrne, D., Jazwinski, C., DeNinno, J, & Fisher, W.A. (1977) Negative sexual attitudes and contraception, in D.Byrne & L.A. Byrne (Eds.) *Exploring human sexuality* (pp. 246-261), New York, Harper & Row.

Cantor, J.R., Zillmann, D., & Bryant, J. (1975) Enhancement of experienced sexual arousal in response to erotic stimuli through misattribution of unrelated residual excitation, Journal of Personality and Social Psychology, 32, 69-75.

Cohen, H.D., Rosen, R.C. & Goldstein, L. (1976) Electroencephalographic laterality changes during human sexual orgasm, Archives of Sexual Behavior, 5, 189-199.

Davidson, J.M. (1980) The psychobiology of sexual experience, in J.M. Davidson & R.J. Davidson (Eds), *The psychobiology of consciousness* (pp.271-332), New York, Plenum Press.

Durden-Smith, J. and D. DiSimone (1983) *Sex and the Brain*, Warner Books, New York.

Fakras, G.M., Sine, L.F., & Evans, I.M. (1979) The effects of distraction, performance demand, stimulus explicitness, and personality on objective and subjective measures of male sexual arousal, Behavior Research and Therapy, 17, 25-32.

Geer, J.H., & Fuhr, R.(1976) Cognitive factors in sexual arousal: The role of distraction, Journal of Consulting and Clinical Psychology, 44, 238-243.

Gellhorn, E.(1957) *Autonomic imbalance and the hypothalamus*, Minneapolis:University of Minnesota Press.

Goldstein, L. (1975) *Time domain analysis of the EEG: The integrative method*, in G. Dolce & H. Kunkel (Eds) CEAN-Computerized EEG analysis (pp.251-270), Stuttgart: Gustav Fisher.

Graber, B., Rohrbaugh, J.W., Newlin, D.B., Varner, J.L., & Ellingson, R.J. (1985) *EEG during masturbation and ejaculation*, Archives of Sexual Behavior, 14, 491-503.

Hubey, H.M. (1999a) Evolution of Intelligence, Kybernetes: International Journal of Systems and Cybernetics, Vol 31, No. 3/4, 2002.

Hubey, H.M. (1999b) *The Diagonal Infinity: Problems of Multiple Scales*, World Scientific, Singapore.

Hubey, H.M. (1991) Catastrophe Theory and Human Sexual Response, presented at the *Third International Symposium on Systems Research, Informatics, & Cybernetics*, Baden-Baden, Germany, August 12-18.

Kaplan, H.S. (1977) Hypoactive sexual desire, Journal of Sex and Marital Therapy, 3, 3-9.

Kaplan, H.S. (1979) *Disorders of sexual desire*, New York, Brunner/Mazel.

Karacan, I. (1976) Impotence: Psyche vs soma, Medical World News, 17, 28.

Karacan, I., Aslan, C., & Hirskhowitz, M. (1983) Erectile mechanisms in man, Science, 220, 1080-1082.

Marshall, G.D., & Zimbardo, P.G. (1979) Affective consequences of inadequately explained physiological arousal, Journal of Personality and Social Psychology, 37, 970-988.

Maslach, C. (1979) Negative emotional biasing of unexplained arousal, Journal of Personality and Social Psychology, 37, 953-969.

Masters, W. and V. Johnson (1966) *Human Sexual Response*, Little and Brown..

Moreault, D., & Follingstadt, D.R. (1978) Sexual fantasies of females as a function of sex guilt and experimental response cues, Journal of Consulting and Clinical Psychology, 46, 1385-1393.

Mosher, D.L., & O'Grady, K.E. (1979) Sex guilt, trait anxiety, and females' subjective sexual arousal to erotica, Motivation and Emotion, 3,

235-249.

Mosovich, A., & Tallafero, A. (1954) Studies of EEG and sex function at orgasm, Diseases of the Nervous System, 15, 218-220.

Mould, D.E. (1980) Neuromuscular aspects of women's orgasms, Journal of Sex Research, 16, 193-201..

Robinson, P. (1976) The modernization of sex, New York, Harper & Row.

Rosen, R. (1988) *Patterns of Sexual Arousal: psychological processes and clinical applications*, Guilford Press.

Rosen, R.C.,. & Leiblum, S.R. (1987) Current approaches to the evaluation of sexual desire disorders, Journal of Sex Research, 23, 141-162.

Sarrel, P.M., Foddy, J., & McKinnon, J.B. (1977) Investigation of human sexual response using a casette recorder, Archives of Sexual Behavior, 6, 341-348.

Schachter, S., & Singer, J. (1962) Cognitive, social, and physiological determinants of emotional state, Psychological Review, 69, 379-397.

Sherfey, M.J. (1966) *The nature and evolution of female sexuality*, New York, Random House.

Thom, R. (1975) *Structural Stability and Morphogenesis; an outline of a general theory of models*, W.A. Benjamin, Reading, MA.

Weiss, H.D. (1972) The physiology of human erection, Annals of Internal Medicine, 76, 793-799.

Zeeman, E. (1977) *Catastrophe Theory: Selected Papers 1972-1977*, Addison-Wesley.