A Unified Theory of Chemical Hypersensitivity

Robert N. Taber BSEE, MSEE[1]

This writer became hypersensitive to an isocyanate about fifteen years ago. For many years this was the only chemical I was sensitive to. This hypersensitivity led to pancreatic insufficiency and food allergies of the masked or cyclic type. This was followed by a rapid accumulation of chemical sensitivities to aromatic materials derived from coniferous/petrochemical sources.

Rendered unemployable by the above process, I have had the leisure to read widely in an effort to understand this process of destruction. Having been trained as an electrical engineer, I do not have the regimented mind set of one trained in medicine or biochemistry. As such, I have attacked the problem more from the point of view of a philosopher. My engineering training and 20 years of experience as a practicing engineer have given me a realistic appreciation of the types of processes that God allows. This, in addition to having my own body to observe and experiment with, with the analytic precision of an engineer, has resulted in the following perception of the processes involved. What follows is a top down analysis of what I have learned. This is the reverse of the order of discovery.

I have recently observed that a reaction to exposure to isocyanate consists of two parts. The first part is the known process involving a bronchial spasm, broncharrea, running nose and occasionally ear blockage. This is a chemical specific reaction. The second part of the reaction I call a chemical non-specific reaction or "flush". This nonspecific reaction was first observed as the only reaction to an exposure to the diverse pollution of a Sears Roebuck store. This flush occurred with the proper (for me) time delay from exposure to reaction and contained none of the chemical specific symptoms associated with an isocyanate exposure. I then recognized that this flush always accompanied an isocyanate reaction but could stand alone and was, therefore, a separate phenomenon. The following is my analysis of this chemical non-specific reaction or flush.

There is an enzyme found and generated throughout the body called Superoxide Dismutase (S.O.D.). This enzyme is found in three forms. These forms contain copper, zinc or manganese. This material is particularly effective in passivating free radicals. S.O.D. levels in lung tissue in people living in a "dirty" city like New York are elevated

1. 314 Cross St. Belmont, MA. 02178

compared to the levels found in the lung tissue of people in a "clean" city like San Francisco. The fact that the cancer rates for both these cities are comparable is attributed to the relative S.O.D. levels. The S.O.D. in the lung tissue passivates reactive material inhaled right in the lung tissue and prevents it from passing to the blood (Pass-water). We know that reactive materials destroy enzymes, enzymes generating membranes and inhibit the regeneration of these membranes. Prolonged exposure to excessive levels of highly reactive materials such as isocyanates would materially damage this barrier and allow free radicals to enter the blood. For years I reacted only to an isocyanate. Eventually sensitivity spread rapidly to a wide range of aromatic materials as my threshold was reduced and the chemical non-specific flush appeared. With the destruction of the S.O.D. barrier in the lung, free radicals are readily transferred to the blood. If the free radical count in the blood exceeds some level, i.e. a threshold, the free radical oxidation process would be initiated and rapidly increase the free radical count. Following is a documentation and analysis of a free standing non-specific flush. This flush occurred several months after I learned that a number of dietary supplements acted as free radical scavengers and antioxidants (Pearson and Shaw, 1982). I increased the magnitude of or added these materials to a supplement regimen based solely on nutrition. The immediate effect of this was to increase my threshold and increase the time delay from exposure to reaction.

The following is a discussion of FIGURE 1.

TIME VARIANT EFFECTS

Delay Duration

The delay part of a reaction has fascinated me for many years. Initially this delay was in excess of one week. Over the years, this gradually shortened to days and then hours. For the past year, the delay was stabilized at from four to six hours. After a significant increase in free radical scavenger and antioxidant supplements, this delay increased five fold to twenty four to twenty eight hours. In addition, the threshold level has increased significantly. The supplements are almost as effective as a gas mask implant. It is clear that in a non-hypersensitive person inhaling a quantity of free radicals, that the quantity in the blood would rapidly decay and not cause a problem. In a hypersensitive person, from the point in time of inhalation of the offensive radicals above the threshold level, the number of radicals in the blood increases. The slope of the increase, at first imperceptable, increases at an increasing rate. Since a free radical propagation process of unlimited capability is being played off against a finite quantity of free radical scavengers and anti-oxidants, these materials must inevitably be used up. This event defines the limit of the delay duration.

Rapid Propagation

With the disappearance of the limiting materials, the free radical propagation cycle is essentially uninhibited. Ample oxygen is available and the result is a very rapid increase in the radical count in the blood.

R:R and ROOH Passivation

Throughout all time, these two processes are in effect. The effectiveness of the R:R termination process is insignificant in low densities of free radicals. The ROOH termination process requires four reactions to effect the termination of one free radical. The propagation cycle is completed in two reactions and produces two new free radicals. The ROOH termination process could only be expected to terminate at best, one out of eight radicals generated by a free swinging propagation reaction. For low levels of radical concentrations, the sum of the effects of both termination processes does not impede the propagation process.

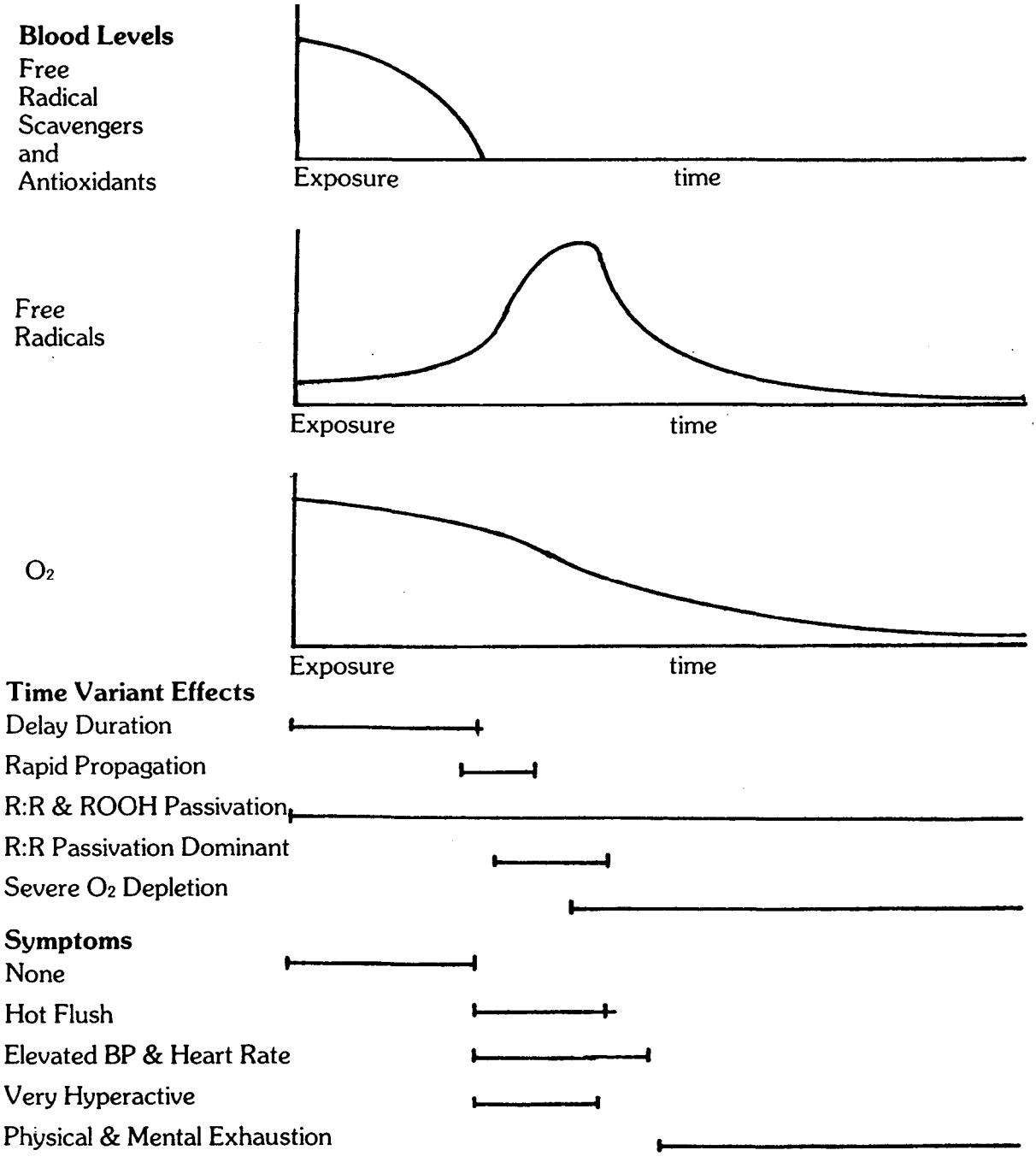
R:R Passivation Dominant

As the density of free radicals in the blood becomes high, the R:R rate of passivation becomes significant, both because the statistical incidence of reactions increases and because each reaction terminates two free radicals. The effect of this is to cause the rate of rise of the free radical count in the blood to decrease. This effect, in addition to a reduction of the availability of oxygen due to depletion by the propagation cycle, results in an eventual leveling of the free radical count in the blood.

CHEMICAL HYPERSENSITIVITY

The Anatomy of a Chemical Hypersensitive Flush as Deduced by a Victim

Figure 1



Free Radical Oxidation Process

Initiation: RH + O2 — R* + HO2

Propagation: R* + O2 — RO2*

RO2 + RH — R* + ROOH

Termination: R* + R* — R:R

R* + HO2 — ROOH

Severe O2 Depletion

In this final phase, the propagation cycle is still struggling for dominance, but is defeated by lack of oxygen. The radical count in the blood falls below the threshold value and normalcy reigns again.

SYMPTOMS

None

The duration of the delay is entirely asymptomatic.

Hot Flush

The first warning that something is awry is a warm feeling and tightness in the forehead. This starts at the rapid rise of free radical count and slowly fades away. The duration of the hot flush is from three to five hours.

Elevated BP & C

As the radical count rises rapidly, so do the blood pressure and heart rate. In a recent example, blood pressure rose from about 145/90 to 160/110 at an early point and 180/115 at a latter point on the peak. Blood pressure tapers off as the radical count decreases. Heart rate rose from a rest rate of 95-100 to 120-140 for the duration of the peak. All of which is consistent with an increased requirement for oxygen.

Very Hyperactive

As the free radical count rises, a state of hyperactivity is induced. Experience has shown that violent exercise helps. This is consistent in that it helps induce anoxia and foreshortens the duration of the flush.

Physical and Mental Exhaustion

As the hyperactive state diminishes, a state of physical and mental exhaustion ensues. This slowly dissipates over a period of one to two hours.

Normal blood levels for S.O.D. are 50 to 80 mcg/ml. 60 mcg/ml is average. A drop to 50 per cent of this level is considered fatal (Passwater). S.O.D., catalase and glutathione peroxidase are enzymes that break down the superoxide free radical, hydrogen peroxide free radicals and peroxide free radicals (Pearson and Shaw, 1982). These are the byproducts of the free radical oxidation process. The above suggests that an

increase in the blood levels of these three enzymes would alter the chemical nonspecific flush! A tablet is available that contains S.O.D. in all three forms, catalase and glutathione peroxidase (S.O.D.-3, made by makers of KAL, Inc., Canoga Park CA, 01304). The addition of a number of these tablets to my daily supplements has had the following result. A reaction to an isocyanate exposure now consists only of the symptoms of the chemical specific reaction. The chemical non-specific flush does not occur! There is a reduction in the amount of toxic material that I accumulate in the course of a day. This is indicated by my energy level and the darkness of my allergic shiners. I have twice acted as if the above indicated would be beneficial and twice obtained the result predicted, first by adding antioxidant and free radical scavenger supplements and then by adding S.O.D., catalase and glutathione peroxidase.

Short term symptoms associated with the flush not mentioned in the above analysis, starting with or after the flush and lasting for several days are as follows: loss in visual acuity, extreme fatigue and intestinal discomfort due to pancreatic dysfunction.

A concise summary of the long term effects of the repeated excitation of the free radical oxidation process is given in Figure 2 (Pearson and Shaw, 1982). This diagram was designed to illustrate the free radical theory of aging; however, it is equally valid to illustrate the effects of the non-specific phase of a chemical reaction. This effect is premature crippling or death by "natural" causes. It is clear that the primary path that I took was from the free radical smog cloud of the diagram through "membrane damage" to "membrane bound enzyme damage" to "loss of self regulation and self repair" to "reduced biochemical adaptability", the membrane and enzyme damage being primarily in the pancreas. This process nearly resulted in death by starvation.

I will now discuss a process that some doctors use with success and others are horrified by. This process is called "neutralization". This process is merely the injection or ingestion of a carefully adjusted quantity of a material after an exposure to a material inhaled or ingested that creates a reaction. This process cancels or "neutralizes" the

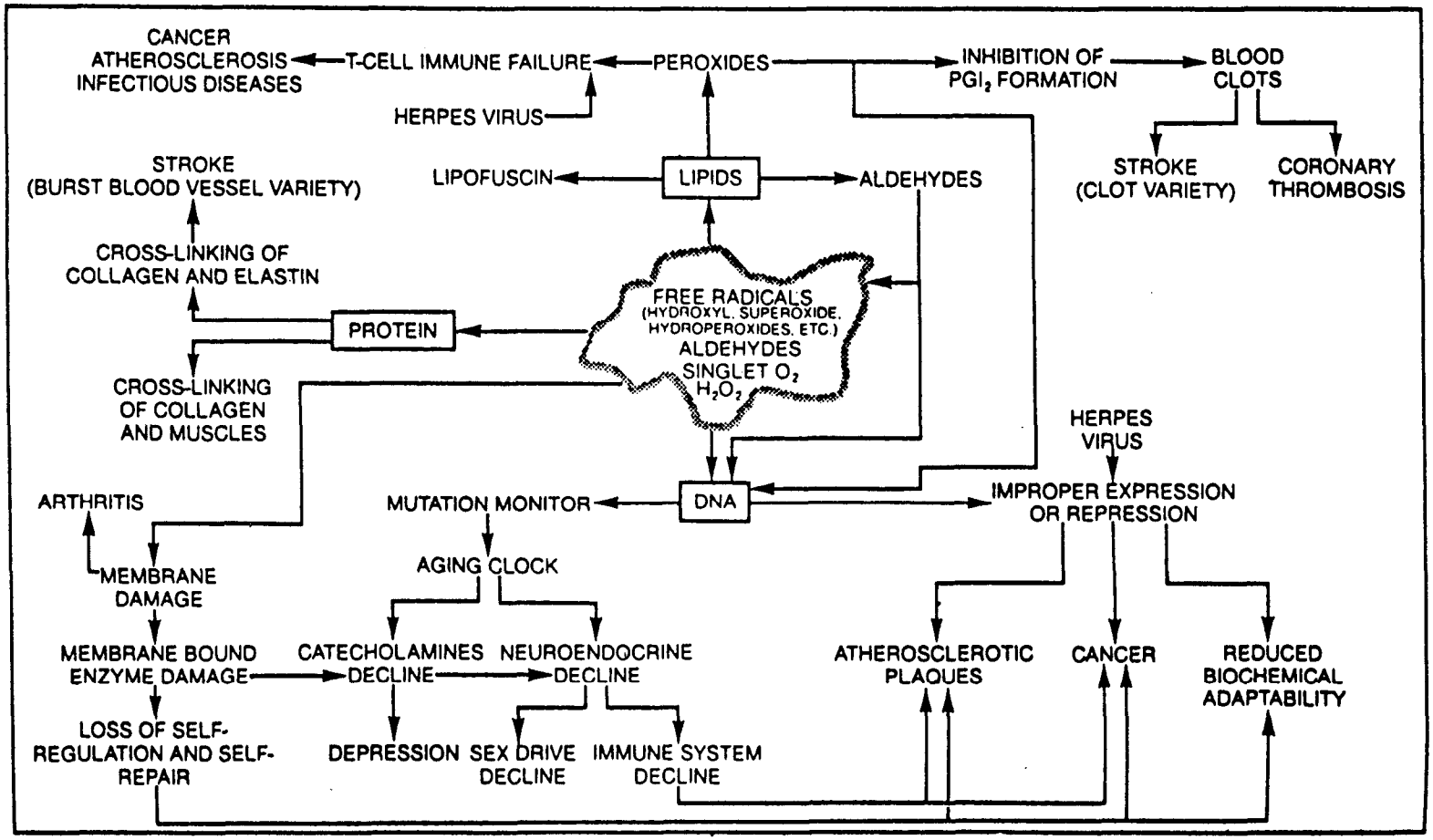


Figure 2 A unified view of aging

From **Life Extension** by Pearson and Shaw, with permission from Warner Books Inc.

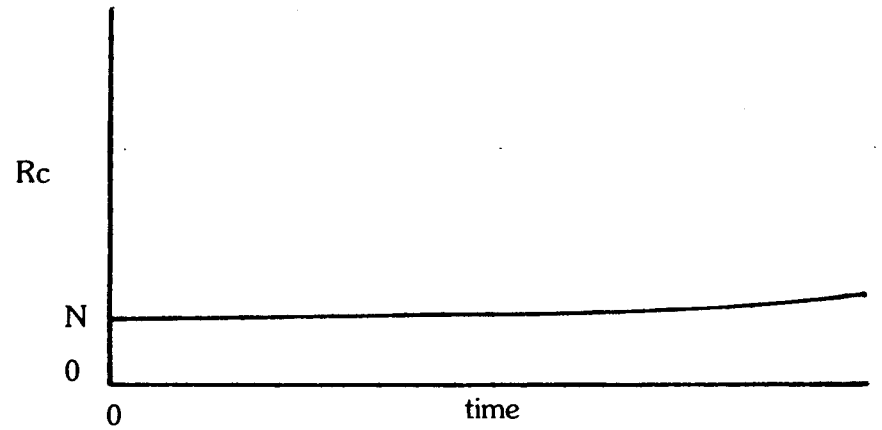
reaction. The material used to effect this neutralization is precisely the same material that would have caused the reaction. On the face of it, this does not make sense. Common sense is a linear process. It cannot predict the results of a non-linear process. In a chemical reaction in a medium, the probability of a reaction occurring is proportional to the density of the ingredients required for the reaction. If one free radical combines with another of the same type to form a more stable compound, then the probability of a reaction occurring is proportional to the square of the number of free radicals of that type present. This is non-linear indeed. It seemed to me that I saw a clue as to why neutralization works in the free radical oxidation relationships and that the solution is analytic.

Following is an attempt to show, in an analytic format, just how this process works. If sufficient data are available, it may also show just how such an analysis might be conducted. I assign a symbol to represent the value of each parameter involved through a sequence of exposure, the delay period during which neutralization is effective, injection of the neutralizing dose and prediction of the radical blood count after neutralization. Figure 3 shows the value of N slowly rising during the delay period after a threshold exceeding exposure at time = 0. Figure 4 displays the increase in probability of a reaction occurring as the blood radical count increases. The probability is proportional to the square of the radical count. Figure 5 is a graphical aid in determination of the value of C. N is the number of free radicals in the blood before neutralization. CN is the number of free radicals in the blood after neutralization. In this outline of a procedure I have used a minimum of symbols for the sake of simplicity. A more elegant outline would normalize values in magnitude, volume and time and be less understandable.

R* + R* — R:R

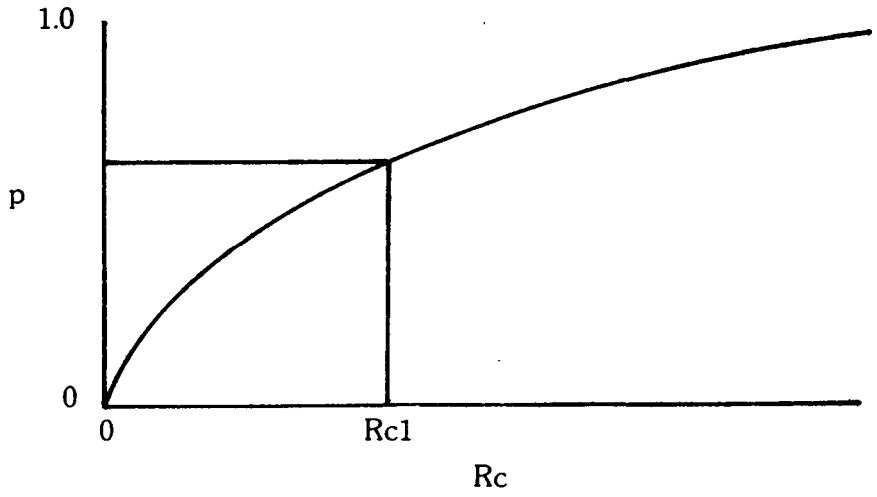
There is leverage in the relationship — 2 free radicals passivated with one reaction!

Figure 3 Rc immediately after a threshold exceeding exposure



Rc = The blood radical count
N = The sensitivity threshold number of radicals
The probability of an R:R passivating reaction increases as the magnitude of Rc increases.

Figure 4 p Vs. Rc



p = The probability of an R:R reaction occurring
n = The number of free radicals passivated in a sample time

n = 2pRc Rl = The number of free radicals injected into the blood (neutralizing dose)

Rcl = The number of free radicals immediately after injection Rcl - Rl = N

Rc2 = The number of free radicals a short time after injection

Rc2 = Rl + N-n

= Rl + N - 2pRcl

= Rl + N - 2p(Rl + N)

Rc2 = (Rl + N)(l - 2p)

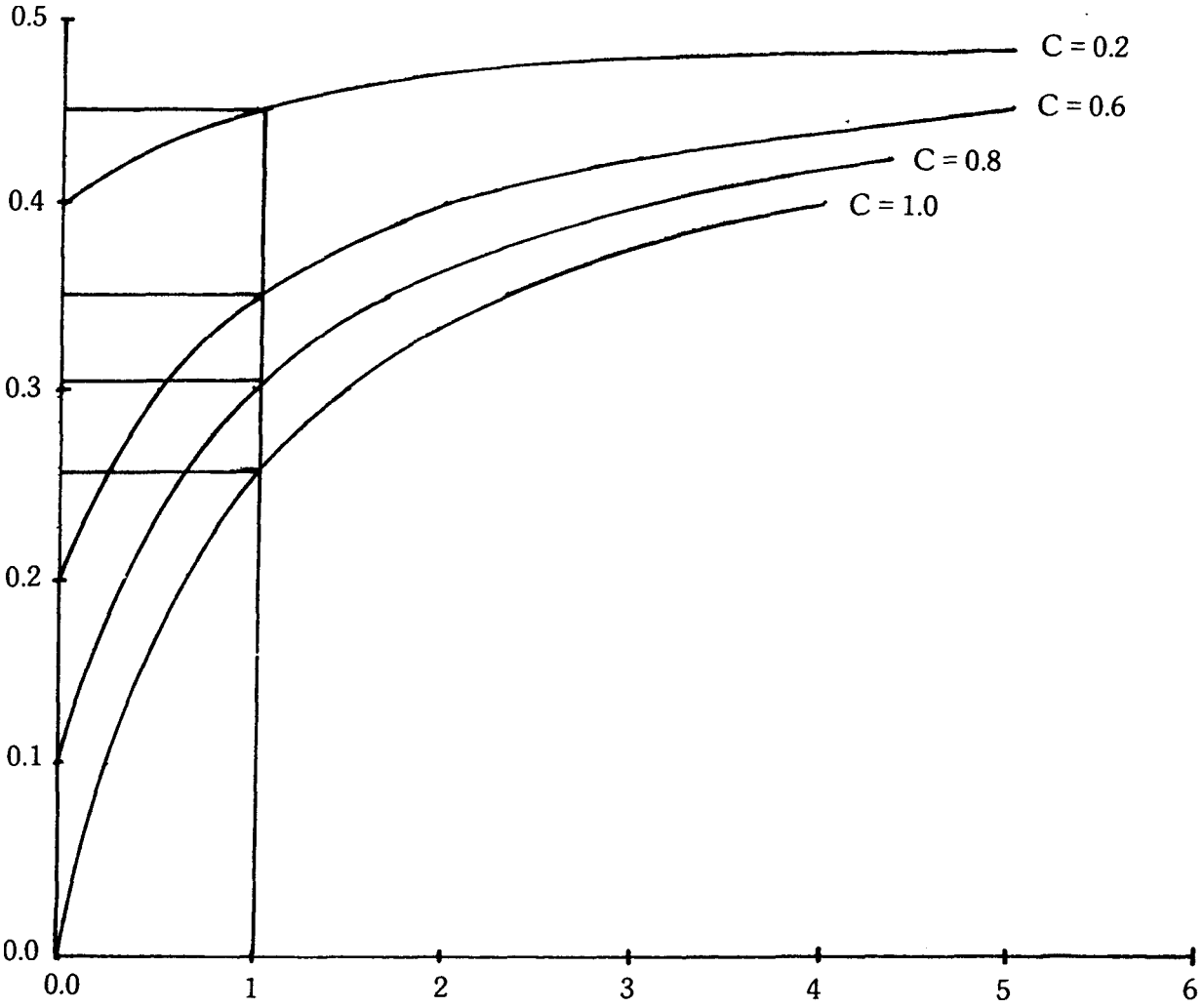
C = A constant of value between 0 and 1

For Rc2 < CN This is required for the blood radical count after injection to be less than before injection

(Rl + N)(l - 2p) < CN Rl - 2pRl + N -
2pN < CN Rl(l - 2p)< CN - N + 2pN
Rl<N(C + 2p-l) (1-2p)

A plot of p Vs. (C + 2p - 1) is given in Figure 5. 1-2 p

Figure 5 Graphic aid for determination of C



$$\frac{(C + 2p - 1)}{(1 - 2p)}$$

If a value for R1 is chosen according to:

$$R1 = \frac{N(C + 2p - 1)}{(1 - 2p)}$$

and a value of p is taken from Figure 5 for a given value of C and (C + 2p -1)/(1 - 2p), and is equal to the value of p taken from Figure 4 using a value of Rc equal to Rcl, then neutralization will have been effected to the value of C that sets the two values of p equal. Example: if C = 0.3, there will be 30 per cent of the number of free radicals in the blood before neutralization, after neutralization.

If the above understanding of the neutralization process is correct, and neutralization works with food allergies, (which it does) then food allergies are merely excitations of the free radical oxidation process with access to the blood through the intestine wall rather than the lung. A masked or cyclic food allergy is, then, one that is being constantly neutralized by frequent ingestion of that food. In me, the chemical non-specific flush and a food reaction are one and the same!

References

PASSWATER, R.A.: Health Food Store Handout, S.O.D., (Superoxide Dismutase). PEARSON, D. and SHAW, S.: Life Extension, p. 483, p. 103, p. 156. Warner Books, Inc., 1982.