was decreased in roots and leaves but was not altered in stems and fruits when the concentration of Be increased in these plant parts.

Beryllium might become injurious to higher plants if high levels were dispersed onto the soil or into ground and irrigation waters where plant roots could come in contact with Be concentrations in excess of 1 ppm in the soil solution. Inhibiting effects of Be on plant growth appear to be centered primarily in the root tissues, and the evidence (*3*) now indicates that Be may inhibit the normal functions of the plant phosphatase enzyme systems. An inhibition of the normal metabolism of inorganic P which the plant attempts to overcome by increased P uptake might account for the increased P concentrations at the higher Be treatments. Apparently Be does not inhibit the formation and functions of chlorophyll (*4*).

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Plasminogen Activity of Plasma and Serum

Abstract. Human plasma and serum were obtained from blood of resting persons. Plasminogen was determined by activation with the optimal amount of streptokinase. As in the majority of cases no significant difference was found between the proteolytic activity of plasma and serum, a blood clot cannot contain more plasminogen than that present in the volume of serum included.

According to current views, a precursor (profibrinolysin, plasminogen) present in plasma reacts with an activator or kinase to produce an enzyme (fibrinolysin, plasmin). The enzyme is inactivated by antifibrinolysins (*1*). The concentration of plasminogen in serum will be further reduced by any capacity of the blood clot to adsorb plasminogen. Therefore, the concentration of plasminogen in plasma should be higher than in serum if these conditions prevail.

In order to obtain information about the difference of plasminogen activity in the two fluids, blood specimens were taken at rest 2 hours after a fat-free breakfast from 14 healthy laboratory personnel who are accustomed to handling blood and do not suffer from any apprehension which might enhance fibrinolysis (*2*). In addition, blood from five patients who were suspected of deficient hemostasis was tested. Some subjects were examined twice.

The first 20 ml of venous blood was placed in two tubes, which were then stoppered and incubated at 37°C for 2 hours. To each milliliter of serum obtained after centrifugation was added 0.02 ml of 1.3*M* tertiary sodium citrate. A further 20 ml of blood was mixed with 0.2 ml of 1.3*M* tertiary sodium citrate in tubes chilled to 2°C.

Plasmin antagonists in cell-free plasma and serum were destroyed by adjusting *p*H to 2.0 and allowing to stand at room temperature for 15 to 30 min (*3*). After neutralization, plasminogen was activated with optimal amounts of streptokinase (Varidase, Lederle). Proteolytic activity was measured after addition of casein (*4*). The results are given in Table 1.

It was calculated from 17 duplicate estimations that the results of plasminogen determinations in plasma and serum are not significantly different, at the .05 confidence level, if they lie between 94.6 and 105.7 percent (*5*). A linear relationship was established between proteolytic activity and concentration of plasma and serum down to 50-percent concentration.

Table 1. Comparison of plasminogen activity of human plasma and serum. One volume of inhibitor-free and neutralized plasma or serum, 1 volume of 3-percent casein solution in borate buffer (*p*H 7.3) and 0.1 volume of 0.154*M* sodium chloride containing between 430 and 830 units of streptokinase (Varidase) per milliliter of plasma or serum were incubated at 37°C for 30 min. The reaction was stopped by chilling the tubes to 2°C and addition of 2 volumes of 20-percent trichloroacetic acid (wt/vol). After centrifugation the optical density of the solution was determined at 280 mμ. For each reaction mixture a corresponding blank was prepared at 2°C and its optical density was subtracted from that of the incubated mixture. Human serum of known protein content was used as standard. Figures represent averages of trichloroacetic acid-soluble material expressed as milligrams of protein per milliliter of original plasma or serum.

Number of determinations	Plasminogen activity				$\frac{B \times 100}{A}$	
	Plasma (*A*)		Serum (*B*)			
1	2.16		1.96		90.7	
14	2.50	(2.16 to 3.27)	2.46	(2.09 to 3.10)	98.4	(94.8 to 99.1)
6	1.73	(1.01 to 2.40)	1.79	(1.04 to 2.45)	103.4	(102 to 105.5)
6	1.78	(1.00 to 2.98)	1.96	(1.07 to 3.20)	110.1	(107 to 115)

The potential proteolytic activities of plasma and serum were not significantly different ($P < .05$) in 20 instances. In six instances serum contained a higher plasminogen activity than plasma and in one case serum activity was lower than the plasma activity. No correlation was found between the plasminogen activity and sex or age (17 to 61 years). From these results it is seen that the concentration of plasminogen in serum was in the majority of cases not less than 95 percent of that in plasma and therefore little adsorption of plasminogen had taken place on the blood clot. Plasminogen determinations carried out with normal human plasma before and after defibrination with bovine thrombin (Parke-Davis) gave results similar to those tabulated.

There is justification to assume from experimental evidence that no loss of plasminogen occurred in the chilled plasma during the time of blood collection and determination as a result of plasmin formation due to the possible presence of plasminogen activators. It follows that no fibrinolytic activity was present in the tested specimens.

The tabulated results have a bearing on the treatment of thrombosis by fibrinolytic agents. In mixtures of purified plasminogen and iodinated fibrin (*6*) it has been observed that some plasminogen is adsorbed by fibrin, and from this finding it has been assumed that fibrinolysis in the circulation results from the activation of plasminogen supposedly present in a thrombus. Also, it has been estimated that approximately 30 percent of the available plasminogen in plasma is adsorbed onto fibrin during clot formation (*7*).

The data presented, however, indicate that a clot prepared from normal blood taken during resting contains no more plasminogen than is present in the

serum included by the clot and that less than 5 percent of plasma plasminogen is adsorbed by the clot. The results suggest that for therapeutic fibrinolysis the concentration of the agent intended to dissolve fibrin should be present in adequate concentration in plasma (*8*).

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Pain Threshold and Discrimination of Pain Intensity during Brief Exposure to Intense Noise

Abstract. Neither pain threshold nor the capacity to discriminate intensity of mild pain was significantly altered during simultaneous stimulation by intense "white" noise. These observations indicate that the reported clinical usefulness of such auditory stimulation during painful therapeutic procedures does not result from an alteration in the capacity to perceive pain.

Gardner *et al.* (*1, 2*) reported that appropriate use of apparatus which simultaneously presents stereophonic music and "white" noise is strikingly effective in reducing discomfort and need for local anesthesia during a variety of therapeutic procedures on the teeth, including extractions. The importance of relaxation, conditioning, and suggestion in modifying painful sensations and reactions is well recognized (*3*), and it has been reported that simultaneous presentation of an intense (140 db) 1000-cy/sec tone raised the pain threshold slightly, apparently by impairing the discrimination capacity of the observer through diversion of his attention (*4*). However, Gardner *et al.* concluded that stimulation with white noise was more effective in reducing pain than would be predicted solely as a consequence of these factors, and that "the noise directly suppresses pain" (*1*). They postulated that the massive barrage of impulses evoked in the auditory pathways by white noise may inhibit central neural aggregates concerned with pain, perhaps through interaction at the level of the reticular formation and thalamus (*2*).

To begin analysis of the mechanisms of this altered response to noxious stimulation, peripheral pain thresholds and discrimination of the intensity of mildly painful stimuli with and without simultaneous brief stimulation with intense white noise were contrasted.

Radiant energy was applied for 3 seconds to skin blackened with India ink at an intensity of from 120 to 240 millicalories per second per square centimeter. During a preliminary instruction period a painful stimulus was defined for the subjects, by demonstration, as one in which pricking pain was barely perceptible in the final instant of a 3-second stimulation. Room temperature was maintained between 22° and 25°C. Eighty stimuli, each to a different area of blackened skin on the volar surface of the forearm, were then successively applied by one investigator. The intensity of each stimulus was determined by another investigator who selected at random from a group of cards representing values from 120 to 240 millicalories. Neither the investigator applying the stimuli nor the subject was informed of the intensity of the stimuli. The range of intensities was selected to contain approximately as many stimuli above as below the pain threshold predicted from previous experiments. There were five cards for 120 mcal, five for 135, five for 150, and so forth. The subject's response to each stimulus was recorded as positive or negative for perceiving pain.

Every other thermal stimulation was accompanied with simultaneous stimulation by noise produced by a Grason Stadler 901-A generator with attenuation above 10,000 cy/sec at an intensity of 120 db relative to 2×10^{-4} dyne/cm^2 presented bilaterally through Permoflux PDR-8 earphones. The responses of a group of ten healthy medical students were ascertained. The group pain threshold was defined as the intensity at which pain was reported from 50 percent of stimuli. The threshold for this group (180 mcal/sec per square centimeter) agrees well with the thresholds reported previously from this laboratory (*5*). No significant

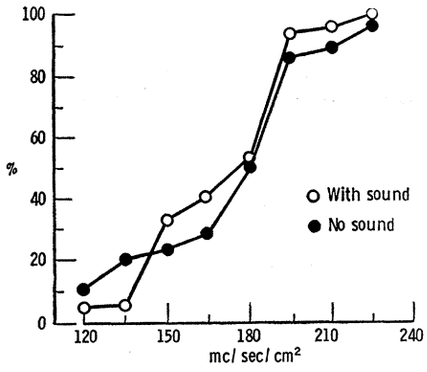

Fig. 1. Percentage of responses indicating that pain was perceived at various intensities of thermal stimulation. For each intensity five responses by each of ten subjects were ascertained. The group pain threshold is the intensity at which pain was reported from 50 percent of stimuli.

alteration in pain threshold as ascertained in this way was induced by simultaneous exposure to intense noise (Fig. 1).

To ascertain the effect of noise on discrimination of the intensity of mildly painful stimuli, pairs of blackened spots were placed on the forehead, the forearm, and the dorsal surface of the hand. The subject was asked to report whether a stimulus presented simultaneously with noise was more or less painful than a standard comparison stimulus presented without noise.

One spot in each pair was exposed to a standard stimulus of 235 mcal/sec per square centimeter without sound. The adjacent spot was then stimulated at an intensity selected at random from a fixed population ranging from 220

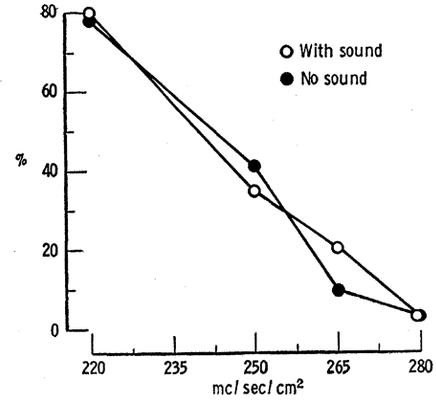

Fig. 2. Percentage of stimuli judged to be less painful than a comparison stimulus of 235 mcal/sec per square centimeter. Stimulation at this intensity was reported to be mildly painful by all subjects. For each intensity five responses by each of ten subjects were ascertained.