J. Physiol. (1966), **187**, *pp.* 35–49
With 10 *text-figures*
Printed in Great Britain

A VISCO-ELASTIC THEORY OF MECHANORECEPTOR ADAPTATION

BY W. T. CATTON AND N. PETOE
From the Department of Physiology, The Medical School, University of Newcastle-upon-Tyne

(*Received* 25 *November* 1965)

SUMMARY

1. Physical analysis of two visco-elastic models was performed, to afford a quantitative basis for examination of a theory of slip as applied to mechanoreceptor adaptation. In one model the coupling force between skin tissue and receptor was considered to be purely viscous; in another it was supposed to consist of parallel viscous and elastic forces, representing the properties of a gel.

2. Predictions from the models were compared with experimental results from frog and rat skin receptors. Good fits with slope–latency and slope–amplitude curves were obtained, with the adjustment of two constants.

3. The excitability changes during long subliminal stimuli showed dynamic and static phases, which developed at different rates as stimulus strength was increased. This behaviour could be explained qualitatively by the more complex model, but quantitative comparisons could not be achieved.

4. Treatment of the skin with tissue-destroying enzymes caused changes in stimulus-response relationships consistent with predictions from the models. The effect of the enzymes seemed to be largely on the elastic coupling forces.

5. The visco-elastic model offers a satisfactory but not exclusive explanation of certain time- and amplitude-dependent features of mechanoreceptor behaviour and also accounts for a specific delay in mechanical excitation.

INTRODUCTION

Evidence is accumulating that adaptation in mechanoreceptors may be attributed in part to mechanical factors (e.g. Eyzaguirre & Kuffler, 1955; Loewenstein, 1956; Hubbard, 1958; Lippold, Nicholls & Redfearn, 1960; Loewenstein & Mendelson, 1965; Oseki & Sato, 1964; Loewenstein & Skalak, 1966). The sensory terminal is considered to be capable of some

degree of slip with respect to the surrounding tissue, the amount of slip being less for a slow adapting than for a rapidly adapting receptor. The coupling forces are likely to include both viscous and elastic components, these being combined in the gel-like mechanical properties of protoplasmic material. With a high elastic modulus little slip can occur and the terminal follows the applied deflexion, so that adaptation is slow. When the modulus is low the main force acting on the terminal is viscous drag and rapid adaptation occurs, since the terminal quickly recovers at the end of a dynamic stimulus, owing to its own elastic properties. Viscous coupling imparts the property of dependence on stimulus velocity, and predicts variation of response latency with velocity and the existence of a critical slope. Elastic coupling would give rise to a discharge under static deflexion, dependent only on stimulus amplitude, and predicts an amplitude threshold. A combination of the two types of force could account for the dynamic and static phases of response characteristic of many receptors.

In a previous paper (Catton, 1966) it was shown that delay occurs in mechanical excitation, which is dependent on stimulus amplitude and duration. In the present paper the properties of two visco-elastic models will be analysed, the predictions from which will be compared with experimental data. Treatment of the skin with enzymes will be used as a test for the theory. Preliminary accounts have already appeared (Catton & PeToe, 1964, 1965).

METHODS

Dorsal skin-nerve preparations from the frog were mounted in a chamber as described in a previous paper (Catton, 1966). Stimuli were applied to the outer skin surface by means of a fine-tipped non-penetrating stylus attached to a moving coil unit. The wave forms available were: (i) linear slope terminating in a plateau; slope, height and plateau duration being adjustable; (ii) rectangular, with duration up to 120 msec. Wave forms were monitored by a variable capacity element mounted on the coil, connected to a discriminator circuit whose output was fed to the second beam of the oscilloscope. Stylus deflexion was calibrated by eyepiece micrometer. Isolation of single receptors was achieved by localization of the stimulus and records were made from the whole nerve. For following excitability changes during long conditioning pulses a second brief test pulse was added on to the conditioning pulse and its amplitude measured directly on the screen.

Enzyme preparations. The enzymes used were hyaluronidase ('Hyalase', Fison's Ltd.); collagenase, Kodak Ltd, and trypsin, BDH. They were dissolved in frog Ringer at pH 7·2, and were applied to the outer skin surface in a Vaselined Perspex retaining ring. Portions of skin from the treated area were removed at various times for histological examination.

RESULTS

Theory of visco-elastic models

Two models were analysed, with the simplest possible assumptions. In the first the coupling medium was assumed to be a viscous fluid and in the second a gel, i.e. a material having both viscous and elastic properties.

The receptor terminal was considered to be stretched by the stimulus, whatever its nature.

The simple series visco-elastic model. Stretching of the terminal occurs as a result of skin deformation and the factors considered were: (i) velocity of skin surface movement, $\dot{x}$; (ii) coefficient of viscosity (η) of the material lying between terminal and surrounding tissue; (iii) resulting extension of the terminal, S; (iv) elastic modulus of the terminal, Y. An arbitrary point of fixation of the proximal zone of the terminal is assumed, extension occurring only in the part distal to this zone (see Fig. 1). Conventional analysis of this model yields the following equations. For constant velocity of displacement

$$S = \tau\dot{x}(1-e^{-t/\tau}). \tag{1}$$

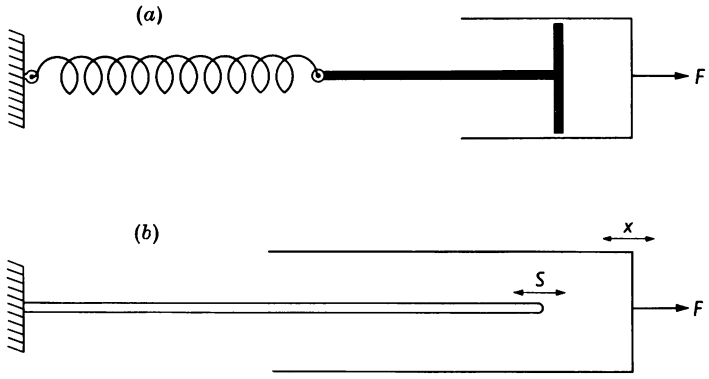

Fig. 1. (*a*) Spring and dashpot model. A force F moves the dashpot at velocity $\dot{x}$, through a displacement x, so that the spring is stretched by an amount S. (*b*) The concept as applied to receptor terminal in skin.

If the threshold extension for initiation of a spike be S' and the latency t', then

$$\dot{x} = \frac{S'}{\tau(1-e^{-t'/\tau})} \tag{2}$$

In these equations τ is a lumped time contant, depending on the viscous coefficient of the material around the fibre, on Young's modulus for the fibre and on geometrical factors. Equation (1) predicts that, for a given velocity, a maximum extension S is reached at $t = \infty$, when $S = \tau\dot{x}$, i.e. the final extension is proportional to stimulus velocity. Figure 2*a* shows plots of the equation for a range of velocities.

Degree of slip. If a straight line representing stimulus gradient is drawn for any curve in Fig. 2*a* the amount of slip at any time is read as the vertical intercept between this line and the S, t graph. Slip increases with time, as represented by plotting $(x-S)$ against time (Fig. 2*b*).

Theoretical slope–latency curves. These are plots of equation (2) as shown

in Fig. 3 for two values of S and τ. The curves show a slope threshold, achieved at $t = \infty$, corresponding to the curve in Fig. 2*a* in which there is an asymptotic approach to the threshold (S') line. The critical slope, $\dot{x}_c$, equals S'/τ. The effects of S' and τ on curve shape were examined on a series of solutions of eqn. (2) obtained from a computer. It was found that τ affected chiefly the 'tail' of the curve (Fig. 3*a*) while S' affected the initial steep section (Fig. 3*b*). It was thus possible to predict what adjustments to make to values of constants in attempting to fit a given experimental curve.

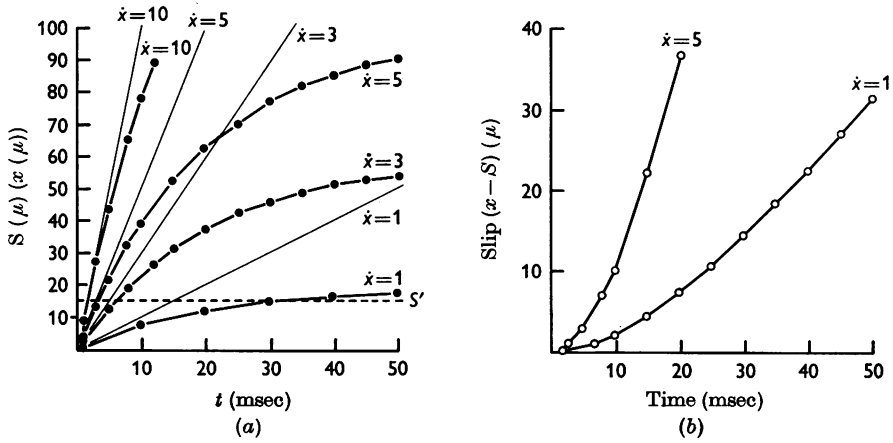

Fig. 2. (*a*) Curves drawn from equation (1) relating predicted extension of terminal (S) with lapse of time during a linearly rising stimulus applied to the skin for velocities ($\dot{x}$) of 1, 3, 5 and 10 μ/msec as shown, τ = 20 msec. On the same ordinate scale are plotted values of stimulus amplitude (x), and the straight lines are drawn for the same velocities as above, to represent the time course of applied displacement. The threshold extension of the terminal (S') is taken arbitrarily as 15 μ (interrupted line), and an impulse would be initiated at that instant where the curve for each velocity crosses this line. The vertical intercept between the x, t and S, t curves gives the absolute slip ($x-S$) at any given time. (*b*) Curves showing predicted increase of slip with time for two values of velocity ($\dot{x}$). τ = 20 msec.

Time constant for delay in excitation. Since near the critical slope the spike latency was very variable, the relation between $\dot{x}$ and t' was better characterized by taking an arbitrary time constant t_c, defined as the abscissal value corresponding to the ordinate $2\dot{x}_c$. Simple calculation shows that $t_c = 0{\cdot}693\tau$.

Theoretical slope–amplitude curve. This was derived graphically from a family of S, t curves (cf. Fig. 2*a*), by taking values of x on the x, t curve corresponding to the time at which the S, t curve crossed the threshold (S') at each velocity. Curves derived in this way are shown in Fig. 5. Each is asymptotic at high slopes to a minimum amplitude (critical

amplitude x_c) and at low slopes to a critical slope $\dot{x}_c$. The critical amplitude represents a value of x such that S' is achieved with minimum latency. The critical slope represents a value of $\dot{x}$ such that S' is only achieved after a long delay.

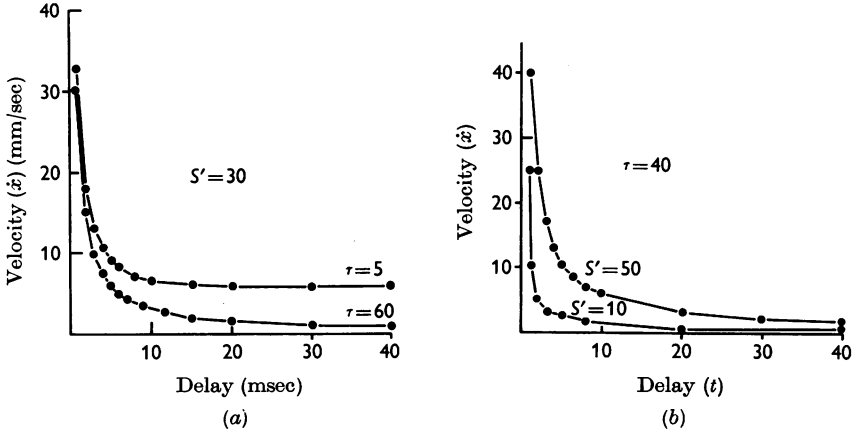

Fig. 3. Curves from eqn. (2); (*a*) with $S' = 30\ \mu$ and $\tau = 5$, $\tau = 60$ msec; (*b*) with $\tau = 40$ msec and $S' = 10$ and $50\ \mu$.

The case of mixed viscous and elastic coupling—the gel medium. The extra factor considered here is the bulk elastic modulus of the gel, G. The derived equation, corresponding to (1) above, is

$$S = K\dot{x}\left[\tau - \frac{\tau^2}{\tau'}\right][1 - e^{-t/\tau}] + Kx\tau/\tau'. \tag{1'}$$

This differs from that for the viscous model (1) in that it contains an additional time constant, τ', and consists of two terms. The first is dependent on velocity, the second on amplitude. τ' is an inverse function of the bulk elastic modulus of the gel. At threshold $S = S'$ when $t = t'$, giving the slope–latency relation

$$\dot{x} = \frac{S'}{K\tau[1-(\tau/\tau')][1-e^{-t'/\tau}] + Kx(\tau/\tau')}. \tag{2'}$$

The response to a step function. Following a step change, as at the beginning of a rectangular pulse, the terminal will undergo relaxation to a new steady length. This is expressed for the gel model by

$$S = S_1 + [\tau'/\tau - 1]S_1 e^{-t/\tau}, \tag{3}$$

where S_1 is the plateau level, approached along the exponential shown by the second term. For an initial displacement S_0 the final plateau level S_1 is given by

$$S_1 = S_0\tau/\tau', \quad \text{where} \quad \tau/\tau' = \frac{kG}{k_1 Y + k_2 G}$$

and is thus seen to be a complex function of the ratio of Y (Young's modulus for the terminal) to G (bulk modulus of the gel). When $G \gg Y$ the plateau remains high, and vice versa.

Applicability of the model equations. Whereas it is possible to arrive at reasonable estimates for the two constants S' and τ in eqn. (2) so that quantitative comparison with experimental curves can be made, this is not true for eqn. (2′) due to its greater complexity. Quantitative correlations were therefore attempted only for experiments where velocity was a dominant factor, as in slope–latency and slope–amplitude relations.

Experimental studies on skin receptors and comparison with theory

Stimulus velocity and latency of spike response

Slope–latency curves were plotted for dorsal skin receptors of the frog and plantar skin receptors of the rat, by varying the slope of stimulus and measuring the latency for each slope. In order that the results should correspond with events at the receptor the latencies were corrected for conduction time. This was done by subtracting the value for latency to an electrical pulse, as described in a previous paper (Catton, 1966). Corrections ranged from 1·5 to 3 msec in different experiments.

Correlation of experimental with theoretical curves

In order to fit an experimental curve it was necessary to choose values of S' and τ to substitute in eqn. (2). An approximate value of τ was obtained by first determining the latency time constant, t_c, and then calculating τ. An estimate of S' could be obtained by noting the amplitude threshold at the steepest slope, a condition where slip was expected to be small. Values of τ from 5 to 80 msec and S' from 5 to 30 μM were used in fitting different curves.

In Fig. 4*a*, *b* are shown comparisons of experimental and theoretical curves for a frog receptor (Fig. 4*a*) and a rat receptor (Fig. 4*b*), from which it is clear that the model equation is able to provide a close fit in each case. Computer solutions to equation (2) were used in curve fitting. The frequency distribution of t_c plotted from sixty-six frog receptors was trimodal (Fig. 4*c*), indicating some heterogeneity in the receptor population in this respect. The first two peaks were taken to correspond to the well-known fast- and slow-adapting receptor groups (A_1 and A_2 receptors of Fessard & Segers, 1943; types (*a*) and (*b*) of Catton, 1958).

Slope–amplitude curves and comparison with theory

Slope–amplitude curves for frog receptors were plotted by varying the slope and measuring the threshold amplitude at each slope. It was more

difficult to match individual experimental curves than with the slope–latency series, there being in particular no guide for choosing the value of τ. In Fig. 5 are shown slope–amplitude curves from three frog receptors and alongside each is a theoretical curve, in which S' was chosen to give the best fit in each case. S' values corresponded to the minimum amplitude at the steepest slope and were 8, 12 and 18 μM respectively for the three receptors.

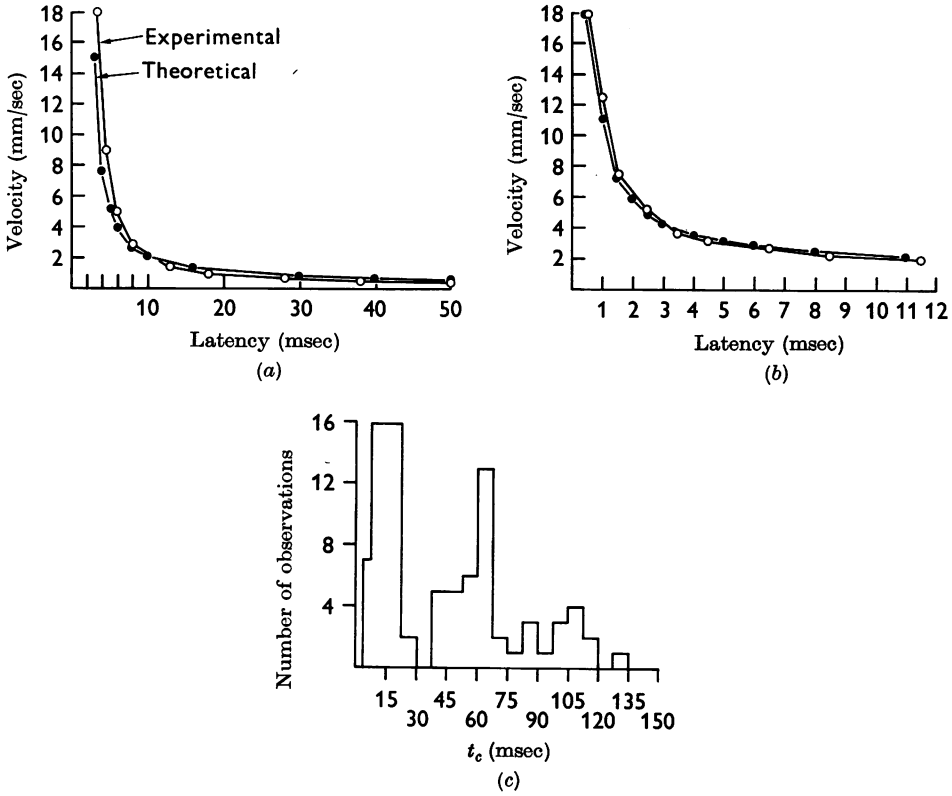

Fig. 4. (*a*) Stimulus slope–latency curve for a frog skin receptor (○) compared with curve (●) drawn from eqn. (2), for $S' = 5\mu$ and $\tau = 40$ msec. (*b*) Stimulus slope–latency curve for a rat skin receptor (○) compared with curve (●) drawn from equation (2) for $S' = 10\,\mu$ and $\tau = 5$ msec. (*c*) Frequency distribution of t_c values for sixty-six different receptors.

Excitability changes during long subliminal stimuli

These were plotted by measuring the amplitude of test pulse which had to be added to the conditioning pulse at various times in order to evoke a threshold response. The over-all changes consisted of dynamic phases at on and off, with a static phase during steady displacement, as shown in

Fig. 6*a* for a range of conditioning pulse amplitudes. In Fig. 6*b* are shown the rates of growth of the dynamic and static phases with increase of conditioning pulse amplitude. The dynamic phase (on) rises almost linearly, whilst the static phase rises slowly and then levels off. Since the rise time of the stimulus was constant, velocity of rise increased linearly

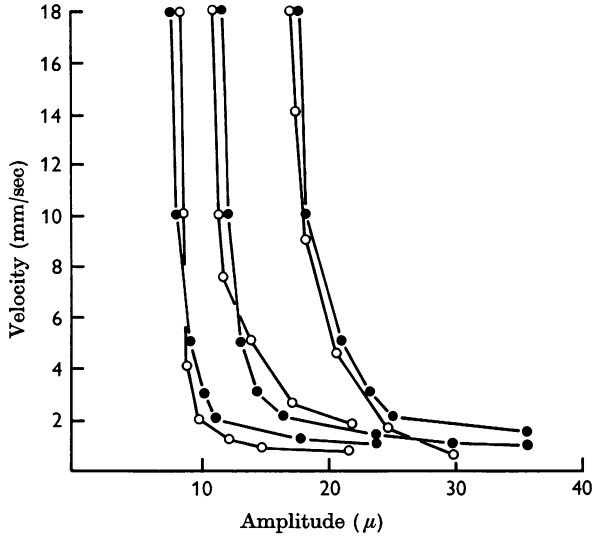

Fig. 5. Experimental slope–amplitude curves (○) for three different frog skin receptors, compared with curves (●) derived from equation (2). The theoretical curves were drawn for a fixed value of τ (20 msec) and values of $S' = 8$, 12 and 18 μ respectively from left to right.

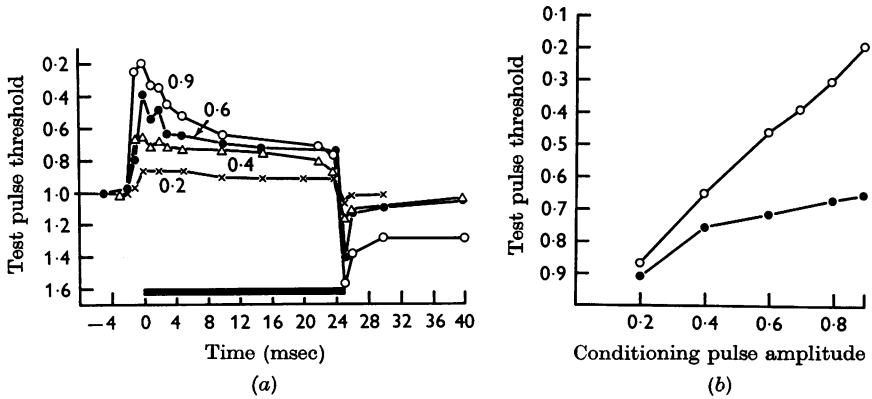

Fig. 6(*a*). Excitability changes during a rectangular conditioning stimulus (horizontal bar) for a given receptor at different amplitudes. Numbers on curves give amplitude of conditioning stimulus in units of threshold. (*b*) Magnitude of dynamic (○) and static (●) phases of response related to amplitude of conditioning rectangular pulse (threshold units).

with amplitude. Thus the rise of excitability in the dynamic phase was proportional to velocity, as predicted by eqn. (1). The static phase commences with a decline from the dynamic peak. Equation (3), derived from the gel model, predicts that this fall should be exponential. A quantitative fit could not be attempted owing to the additional constant τ' which could not be evaluated. The final plateau level (S_1, eqn. (3)) would be complexly dependent on the ratio of the bulk modulus of the gel (G) and Young's modulus of the fibre (Y). If G/Y were large the plateau would be high and vice versa. A range of behaviour was indeed found in different receptors in this respect, as also reported by Lindblom (1963) for toad skin receptors.

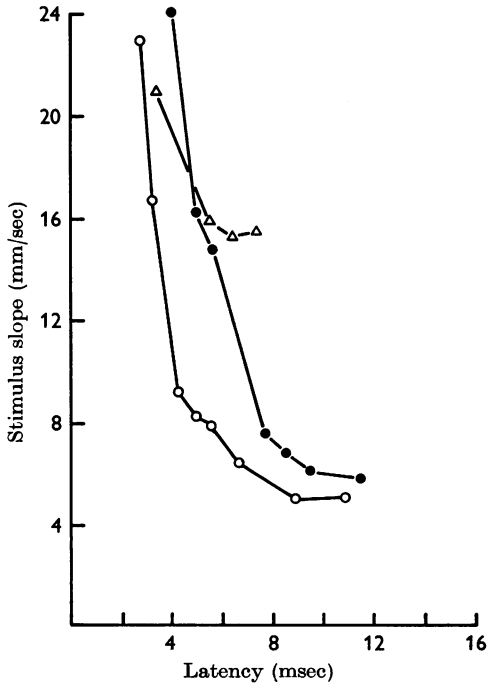

Fig. 7. Slope–latency curves for a frog skin receptor of a type showing high critical slope and short-latency base. ○, control; ●, 30 min and △, 70 min after application of 'Hyalase, 600 i.u./ml. The curve is displaced to the right and the critical slope rises sharply.

Dynamic and static phases of impulse discharge

Whereas all receptors could be excited by rising stimuli, only a small proportion produced a train discharge during steady deformation, and this required an amplitude 5–10 times higher than for excitation by a slope. The discharge behaviour with long supraliminal stimuli thus imaged the time course of excitability changes during subliminal stimulation.

Lindblom (1963, 1965) has reported similar behaviour in toad and monkey receptors.

Tissue-destroying enzymes used to test the validity of the theory

Skin preparations were treated with enzymes expected to alter the mechanical properties, and the responses were measured before and after treatment.

Hyaluronidase. Concentrations used ranged from 300 to 1500 u./ml. and solutions were allowed to act for 0·5–6 hr.

In twelve experiments on the slope–latency curve enzyme treatment caused a shift to the right of the curve in nine cases, a result expected from the theory assuming the enzyme were to loosen the bonding of the sensory terminal so that a higher slope was needed to excite at the same latency. Figure 7 shows for one receptor the effect of 600 i.u./ml. of 'Hyalase'.

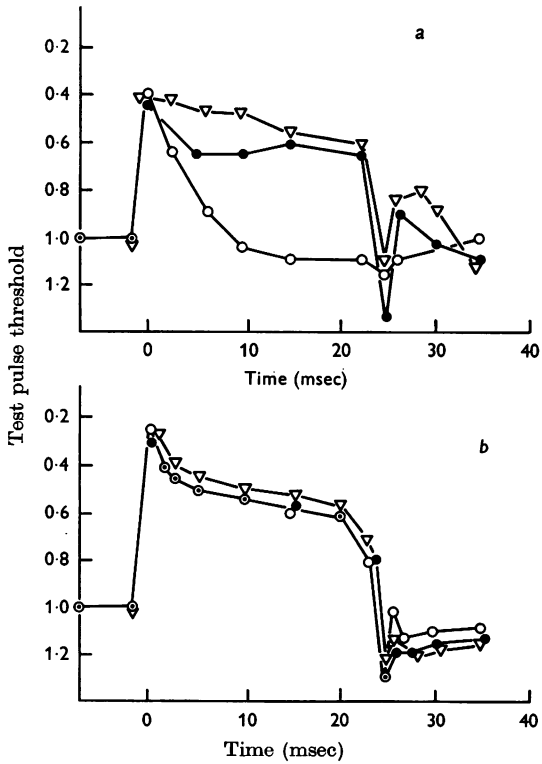

Fig. 8. Excitability changes during a 22 msec rectangular conditioning stimulus (frog skin receptor). (*a*) Control curve (△) and curves plotted after 20 min (●) and 60 min (○) of treatment with 'Hyalase' 600 i.u./ml. (*b*) Control curve (△) and curves (●, ○) after similar periods of treatment with previously boiled enzyme solution.

After 30 min there was a shift to the right and a small increase of critical slope. After 70 min there was a marked rise of critical slope.

In four experiments on the excitability changes during a long conditioning pulse the effect of the enzyme was chiefly on the static phase, a typical result being shown in Fig. 8*a*. After 60 min of treatment the static phase was almost completely abolished, the dynamic phase at 'on' being scarcely affected. When the enzyme solution was previously boiled (Fig. 8*b*) this effect was not seen, confirming that there was an effect specific to the enzyme. Skin sections after treatment showed little change in microscopic structure, except in a few cases where there was partial separation of epidermal cells.

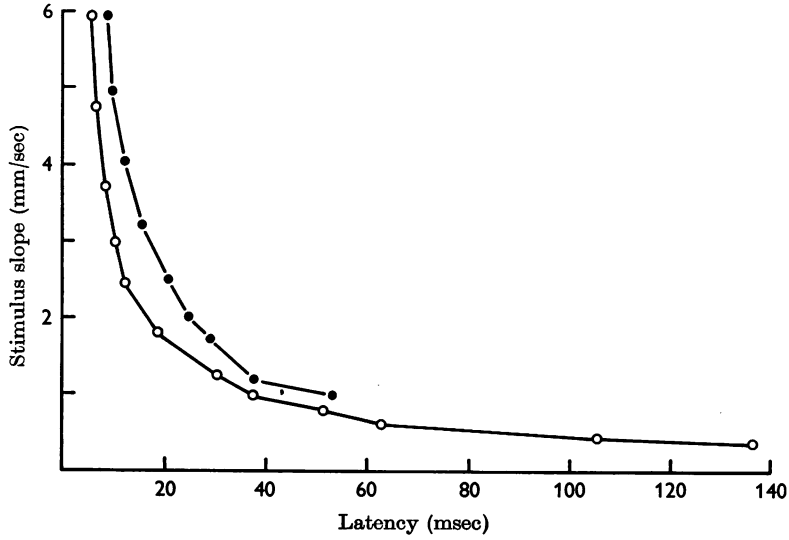

Fig. 9. Slope–latency curves for a frog skin receptor. ○, control; ●, after 2 hr treatment with 800 'C' units of collagenase dissolved in 1 ml. Curve is displaced to right and critical slope has risen.

Collagenase. The enzyme was dissolved in Ringer solution in concentrations from 800 to 80,000 u./ml. In fourteen experiments on the slope–latency curve there was a shift to the right in all cases, in accord with theoretical expectation. Figure 9 shows a result where 800 u. were applied for 2 hr. There was a rise in critical slope and in amplitude threshold. The latter rose so high in some cases that the skin was no longer sensitive to mechanical stimuli but would still respond to electrical stimuli.

In five experiments on excitability changes during a long conditioning pulse the effects were mainly, as with hyaluronidase, on the plateau phase, as shown in Fig. 10. The enzyme produced gross macroscopical changes and after 6–12 hr the skin appeared thin and translucent; the nerves were

floating free and could easily be detached from the skin. Histological controls showed extensive destruction of collagen; after 24 hr treatment with 40,000 u. little but the epidermis remained.

Trypsin. This enzyme was tried because it has been shown to have a marked effect on the gross mechanical properties of some rat tissues (skin and cervix uteri) as reported by Harkness & Harkness (1959). It was applied in 0·2 % solution at pH 7. In six experiments the slope–latency curve was shifted to the right in two cases, to the left in two others and in two cases there was no effect.

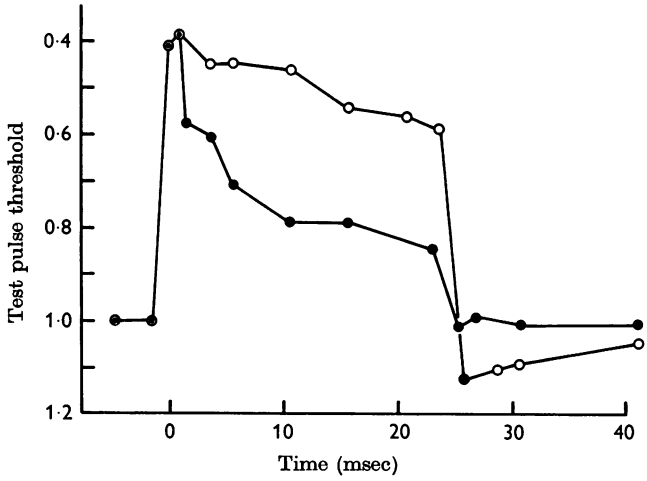

Fig. 10. Excitability changes in a frog skin receptor during a 25 msec conditioning pulse. ○, control; ●, after treatment for 90 min with collagenase 4000 u./ml.

DISCUSSION

An early indication of a possible role of mechanical factors in adaptation was the work of Eyzaguirre & Kuffler (1955) on crayfish stretch receptors. Thus the slowly adapting type showed a maintained receptor potential and was anatomically more firmly bound to the muscle than the slowly adapting type, whose receptor potential decayed rapidly. Loewenstein (1956) offered evidence that the rate of adaptation of frog skin receptors was dependent on static stretch. Hubbard (1958) showed that when a Pacinian corpuscle was compressed and released, only transient movements of the inner core occurred, accounting for the brief receptor potentials evoked in the intact organ. Loewenstein & Mendelson (1965) and Oseki & Sato (1965) have shown that when the lamellae are removed a plateau appears in the receptor potential response. The spike discharge however was only a little extended, i.e. adaptation was occurring also in

the spike-generating mechanism. Loewenstein & Skalak (1966) in an extensive analysis have confirmed that the Pacinian corpuscle is a mechanical filter of high-pass characteristics.

In the slowly adapting muscle spindle Lippold *et al.* (1960) showed that adaptation occurs with mechanical stretch but not with direct current excitation. Brown (1965) has reported similarly for the crayfish slow receptor. Catton (1966), comparing the latencies of skin receptors to electrical and mechanical stimuli, concluded that a delay occurs specifically attributed to the mechanical mode of excitation and that this delay is influenced by the form of the mechanical stimulus.

A feature of many mechanoreceptors is that they can be excited by forces applied in different directions. For example, the receptors in frog skin are excited equally readily by stimuli applied normally or tangentially to the skin, and also give an off-response on decompression. Since the sensory terminals have no uniform orientation, and occur over a range of depths in a non-homogeneous medium, it is not possible to predict what type of deformation may occur with a given stimulus. In the muscle spindle the morphology of the nerve terminal is so complex that it is difficult to predict what change in its conformation might result from simple extension of the intrafusal fibre. Katz (1960) has described fine structure of the spindle terminals which could account for the typical dynamic and static phases of response. The well-defined structure of the Pacinian corpuscle lends itself to analysis, although even here the treatment is necessarily elaborate (Loewenstein & Skalak, 1966). In view of the many uncertainties in the relations of the free nerve terminal in skin the simplest possible assumptions were made in the present work. However, the finding that experimental slope–latency and slope–amplitude curves could be so clearly matched by the visco-elastic model, with reasonable choice of constants, does perhaps no more than show that the experimental curves follow a law of the same general type, which might be due to some other process, e.g. accommodation of the nerve terminal. Indeed Loewenstein & Mendelson (1965) conclude that accommodation must play a part in the adaptation shown by the Pacinian corpuscle, although the mechanical characteristics of the organ also contribute. Specific implication of mechanical factors in the adaptation of skin receptors seems, however, to be indicated by (*a*) the presence of a delay in mechanical excitation (Catton, 1966), and (*b*) the alterations in stimulus–response relation caused by tissue-destroying enzymes. In nine out of twelve experiments hyaluronidase displaced the slope–latency curve to the right, an effect also produced by collagenase in all of fourteen experiments. Hyaluronidase, by its action in loosening intercellular bonds, would be expected to facilitate slippage of those terminals which penetrated between epidermal cells. However, there are

many terminals which end in the deeper layers, and in cases where one of these was stimulated the enzyme might fail to produce an effect. This may have applied in the three cases where there was no change in the slope–latency curve. Since there is no collagen in the epidermis one must assume that the effect of collagenase was due to destruction of collagen in the deeper layers. A thin band of collagen lies immediately under the epidermis, and dissolution of this would allow greater free movement of any terminals embedded in it; but further it would allow freer movement of the proximal zones of fibres which terminated in the epidermis. Loosening of the proximal anchorage would produce an effect on stimulus–response parameters indistinguishable from that of increased slippage of the terminal in the epidermis.

The visco-elastic theory, in its simpler form, assumes that the shape of the slope–latency curve is solely dependent on viscous coupling forces. The changes brought about by the enzymes were, however, generally more marked on the plateau phase of the excitability curves for long stimuli. At low stimulus velocities the contribution of elastic forces to the effective extension of the terminal would be expected to increase relative to the viscous force. The marked rise of critical slope and of amplitude threshold seen in some enzyme experiments could then be attributed largely to weakening of elastic bonding. The shift to the right of the slope–latency curves could be due to an action of the enzymes on both viscous and elastic components, the latter being probably predominant.

This work was assisted by an equipment grant from the Science Research Council.

REFERENCES

BROWN, M. C. (1965). Electrical and mechanical stimulation of slowly adapting receptors in the crayfish. *J. Physiol.* **181**, 1–2*P*.

CATTON, W. T. (1958). Some properties of frog skin mechanoreceptors. *J. Physiol.* **141**, 305–322.

CATTON, W. T. & PETOE, N. (1964). A visco-elastic model for the cutaneous mechanoreceptor. *J. Physiol.* **172**, 21–22*P*.

CATTON, W. T. & PETOE, N. (1965). Mechanoreceptor adaptation: tests of the validity of a visco-elastic model. *J. Physiol.* **179**, 45–46*P*.

CATTON, W. T. (1966). A comparison of the responses of frog skin receptors to mechanical and electrical stimulation. *J. Physiol.* **187**, 23–33.

EYZAGUIRRE, C. & KUFFLER, S. W. (1955). Processes of excitation in the dendrites and soma of single isolated sensory nerve cells of lobster and crayfish. *J. gen. Physiol.* **39**, 87–119.

FESSARD, A. & SEGERS, M. (1943). Quelques charactères différentiels des récepteurs cutanés. *C.r. Séanc. Soc. Biol.* **137**, 212–213.

HARKNESS, M. L. R. & HARKNESS, R. D. (1959). Effects of enzymes on mechanical properties of tissues. *Nature, Lond.* **183**, 1821–1822.

HUBBARD, S. J. (1958). A study of rapid mechanical events in a mechanoreceptor. *J. Physiol.* **141**, 198–218.

KATZ, B. (1960). Sensory terminations in the muscle spindle of the frog. *J. Physiol.* **152**, 13*P*.

LINDBLOM, U. (1963). Phasic and static excitability of touch receptors in toad skin. *Acta physiol. scand.* **59**, 410–423.

LINDBLOM, U. (1965). Properties of touch receptors in distal glabrous skin of the monkey. *J. Neurophysiol.* **28**, 966–985.

LIPPOLD, O. C. J., NICHOLLS, J. G. & REDFEARN, J. W. T. (1960). Electrical and mechanical factors in the adaptation of the mammalian muscle spindle. *J. Physiol.* **153**, 209–217.

LOEWENSTEIN, W. R. (1956). Excitation and changes in adaptation by stretch of mechanoreceptors. *J. Physiol.* **133**, 588–602.

LOEWENSTEIN, W. R. & MENDELSON, M. (1965). Components of receptor adaptation in a Pacinian corpuscle. *J. Physiol.* **177**, 377–397.

LOEWENSTEIN, W. R. & SKALAK, R. (1966). Mechanical transmission in a Pacinian corpuscle. An analysis and a theory. *J. Physiol.* **182**, 346–378.

OSEKI, M. & SATO, M. (1964). Initiation of impulses at the non-myelinated nerve terminal in Pacinian corpuscles. *J. Physiol.* **170**, 167–185.