THE AUSTRALIAN
JOURNAL OF PHYSIOTHERAPY

VOLUME XVIII DECEMBER, 1972 NUMBER 4

WHY DO PHYSICAL TREATMENTS RELIEVE PAIN?[1]

J. M. GANNE, M.C.S.P., DIP.T.P., M.A.P.A.
Senior Lecturer in Physiotherapy, South Australian Institute of Technology

Those engaged in the clinical rather than the experimental field of medicine are constantly striving to improve their methods of relieving pain, using chemotherapy, surgery or physical therapy according to their particular specialization. This is hardly surprising, since all hospital departments, wards and private clinics are filled with a large proportion of patients seeking, primarily, relief from pain.

It is well to remember that this problem of pain, wherever it is and whatever its cause, will usually be accompanied by some interference with function. This may be manifested in many different ways; an impairment in concentration on mental work, a slight decrease in efficiency in performing a physical task, or complete inability to use a joint or a whole limb to carry out one's job.

Interference with function could appear to have an important bearing on the factors responsible for perpetuating sensations of pain once these have been triggered by injury and disease (assuming that the initial injury and stress and pathological processes are not constantly repeated). We are surely familiar with such a situation. An extreme example is worth recalling here to make the point clear. An elderly in-patient was referred for treatment following a cellulitis affecting the lower leg and dorsum of foot. She had been treated, as one would expect, with antibiotics, complete bed rest and a bed cradle (but with no splint). When first examined by the physiotherapist, the infection had resolved and there was very little swelling. There was some discoloration and she kept the leg completely immobile and away from any contact. She complained of constant burning pain and hypersensitivity. She had in fact a good range of movements when these were gently coaxed, but was very apprehensive and wholly preoccupied with her leg.

Simple exercises several times a day, removal of the cradle, wearing slippers and reeducation of walking was all that was needed to cure her pain in a few days.

The mechanisms whereby such a pain pathway is kept active may be very complex but whatever they are, they will be acting on some part of the nervous system, because this is the system which signals pain to our conscious mind.

The lack of movement and function of the part may therefore:

1. Aggravate the local biochemical situation in the tissues around the peripheral sensory nerve endings which are the receptors for pain so that the local factors causing the pain nerve fibres to pick up impulses are unresolved.
2. Disturb the normal transmission of other patterns of afferent stimuli from the local area. These afferent stimuli are coming from stretch receptors in joints and muscles which would be stimulated by normal movement of the joints and muscles. Others come from the normal thermal, tactile, pressure and vibratory stimuli received on the skin or by the subcutaneous tissues when a part of the body is subjected to its usual contacts during function. The spinal cord would be receiving many less messages of this type at the first synaptic level and the pain stimuli could therefore be facilitated by lack of competition. This possible phenomenon will be referred to in more detail later. (The patient referred to in the example was lying with her foot protected from the bed clothes by a cradle).

[1]Presented to the Physiotherapy Society of South Australia, October, 1971. Received July, 1972.

We know well that the perception of these painful sensations can be further influenced by processes of inhibition and facilitation related to psychological phenomena or the influence of mental and emotional processes on the perception of pain, our "state of mind" in lay terms. The patient who has been lying in bed with a painful foot for a few weeks, neither moving nor reading nor occupying her mind in any way has less chance of inhibiting pain circuits.

In contrast, pain impulses are undoubtedly blocked by high emotional states, as in battle and by conditioning. We need to be aware also of the reality of psychosomatic pain so that we do not waste our time treating this type of pain by physical means, and most of us would agree that it is a waste of time. Indeed one could say that one of the characteristics of true psychosomatic pain is that it does not respond to treatment by physical means and so the treatment of this type of pain is not relevant to this paper.

The pain that we treat most commonly is related to disorders of the muscular skeletal system. At times some of the less common abnormal unpleasant sensations which are sequelae to damage of the peripheral nerves come our way, for example, post herpetic pain, post traumatic hyperalgesias, amputation pains and scars. One wishes that the latter group of conditions were referred more often, as the application of cutaneous stimuli are often dramatically effective where other means of treatment fail.

This paper is concerned with considering what we might be doing when we control pain and at what points in the transmission of stimuli, from the periphery to the conscious levels of the central nervous system, physical treatments act.

More information is available now concerning the points in the pathway where analgesic drugs act; for example, salicylates act on the peripheral receptors, morphia acts on the higher levels of the central nervous system. The success of physical treatments, that is movement, passive and active, cold, heat and other counter irritants, is unquestionable in many cases and sufficiently rapid, (that is, at times, immediately effective) so that there is no doubt that the relief has been brought about by the specific treatment and not by the passage of time. Because the mode of action is still open to much speculation there is a tendency for underestimating the value of physical agents as compared with surgery or chemotherapy.

Physiotherapists have nevertheless a golden opportunity of affecting the afferent nervous system precisely, through the application of the different varieties of sensory modalities that the body normally responds to. I mean that we can use stimuli capable of initiating impulses via the many different types of receptors and these impulses can influence the transmission of afferent input to the brain, lowering or raising the thresholds to other stimuli. When we move and stretch joints and muscles we are stimulating mechanical receptors sensitive to touch, pressure, stretch and vibration; cold and heat act on thermoreceptors, low frequency currents are probably acting on the larger touch and pricking pain receptors in the skin, some more noxious stimuli such as cautery are stimulating superficial pain receptors. It is hard to explain the *immediate* relief experienced when intermittent pressure is applied to an aching immobile thoracic joint, or that produced by the sinusoidal current on an acutely tender hypersensitive area, or again, relief within a minute or so when ice is applied to post herpetic pain, other than by a direct effect on nervous pathways. Following these immediate effects we can then postulate that more lasting effects could be obtained through alterations in local metabolism due to changes in local circulation and permeability of vessels and cells which would affect local ionic concentrations and local stasis of fluid. There is no doubt that movement and heat and cold and massage can all bring about a redistribution of local tissue fluids and alterations in circulation.

The most popular theory of the actual mechanism which initiates pain stimuli locally is through the liberation of a chemical pain producing substance. It seems that many authorities are in favour of accepting that pain responses are mediated by chemosensitive pain receptors.

Much experimental work has been carried out with pain producing substances or algesics,

such as some amines (acetylcholine, 5 hydroxytriptamine and histamine) various inflammatory exudates, K-ions and raised and lowered H-ion concentrations also with peptides such as bradykinin. These substances have been applied to blister bases and injected into various parts of the body and have produced pain, without actually damaging the tissues, in arterial walls, in the skin, in viscera and on the peritoneum. Certain ions and peptides present in the body would therefore seem capable of stimulating chemo-sensitive pain receptors under certain conditions. Bradykinin is apparently formed by the action of a specific enzyme which is liberated from tissues or leucocytes when these are suitably provoked and this enzyme acts on substrates in the alpha 2 globulin fraction of plasma (Lim, 1970). Further, bradykinin is readily destroyed by another enzyme present in lymph or circulating cells. Because of this it is tempting to feel that local changes in permeability of cell membranes and circulatory movements could assist in removing or breaking up the pain substance so that it no longer irritates the fine pain nerve endings. Could this effect be an immediate one? It is very doubtful.

Bradykinin and other pain producing substances do not only produce pain but also the other signs of inflammation, capillary vasodilatation and increased permeability with oedema. Bradykinin apparently does not produce pain when injected straight into vascular tissues, for example, below the skin, in the muscles, and intravenously, because it is inactivated by the plasma before it can reach the sensory nerve endings. Nor does it produce pain in the intestinal lumen.

On the other hand the pain evoked by local injection of bradykinin is increased if the part injected is rendered ischaemic. This would seem to follow logically. Lim *(ibid)*, discussing the adequate stimulus for excitation of pain receptors suggests that "excitation may depend upon electrophilic attraction, with the assumption that the receptor sites are anionic or electron rich and that algesic agents, or some part of their molecule are cationic or electron deficient. Electrophilic attraction thus provides the *raison d' être* of the pain receptors if it enables the chemoceptor to signal the increasing concentration of H-ions in ischemia, or the accrual of algesic plasma peptides which herald the onset of inflammation consequent to injury or disease".

If indeed certain concentrations of H and K-ions can cause pain it is interesting to speculate whether ultra-sound and the anodal effect of direct current could alter the distribution of these ions locally. Certainly hydrogen ions are light and are likely to move more easily under the ultra-sound beam or the repelling influence of the positive pole of the anode. One could, I suppose, equally speculate that movement of hydrogen ions stimulates the sensory receptors, or helps to stretch fibrous tissues. Electron microscopy studies have shown that therapeutic doses of ultrasound waves are capable of separating collagen fibres from each other, the effect being due to action on the cement substance between the fibres (Gertsen, 1955).

Perhaps we have tended to overestimate the effects of circulatory changes on pain relief and underestimate the effects of physical stimuli on the temporal and spatial organization of impulses which are relayed to the brain. I will return to this point in more detail.

Let us consider further anatomical and physiological details concerning:

(a) the receptors which we can influence in the skin and deeper tissues;
(b) the fibres by which they transmit sensory modalities to the spinal cord;
(c) the termination of these fibres in the spinal cord and the influence of possible inhibitory and facilitatory mechanisms at the first synapse in the cord.

Although further knowledge is required in relation to the local adequate stimuli for pain and the anatomical pathways for transmission of pain in the central nervous system, considerable information is available on the specifity of different receptors and fibres, or in other words the ability of certain receptors to respond only or more particularly to certain definite stimuli which are transmitted up certain definite fibres, to certain specific areas of the cord and we know that these fibres can be influenced in different ways by certain physical and chemical agents.

However, although it has been possible to isolate potentials in individual fibres resulting from cold or hot or touch stimuli, Brodal (1969) points out that it is necessary to "realize that what is perceived by the mind in normal life is not the stimulus of a single receptor or frequently not only a single type of receptor. What the individual experiences is a complex impression resulting from spatial and temporal summation of stimuli of different kinds".

Cutaneous afferent receptors can be divided into three groups:

1. Mechanical type receptors — Touch, Pressure, Vibration
2. Thermal receptors — Heat, Cold
3. Pain receptors responding to noxious stimuli.

The deeper tissues, including bones and joint structures also have mechanical receptors sensitive to vibration, pressure and stretch which signal movement and position from capsules and ligaments and also pain receptors.

Muscles have three different types of stretch receptors and also pain receptors. Not only are there therefore receptors which will respond more specifically to these different modalities but further study has shown that each type of modality is signalled by more than one type of receptor.

They vary in their rate of adaptability to a particular sensory stimulus and so in their ability to signal a constant discharge related to a steady state, or then a more phasic discharge related to quicker rates of change in state. Also their messages will be transmitted along fibres which vary in diameter and consequently in their rate of transmission to the cord. For instance cold receptors signal temperatures in a range between 10°C and 38°C though their optimum range of operation is between 16°C and 27°C; some fibres respond steadily to a given temperature while others discharge rather when there is a lowering of the skin temperature at that particular spot. Warmth fibres operate maximaly between 38°C and 43° and not below 20°C. Experiments carried out on the tongue showed that finer warmth fibres cease firing at 45°C to 47°C and pain fibres take over transmission of the impulse though there is an initial quick phasic response from warm and cold fibres. Changes in skin temperature of only .2°C are apparently sufficient to cause definite discharges in the thermal fibres. Although specific thermal receptors have not been identified some of the fine free nerve endings are specifically receptive to thermal as well as touch and pain stimuli. There is some overlap so that some receptor units appear to respond to thermal and mechanical stimuli, also the responses of tactile receptors can be influenced by changes in temperature. (Starling and Lovatt Evans, 1968).

We are all familiar with the two types of pain which can clearly be elicited from the skin or in the deeper tissues, a "quick" pricking or stabbing pain and a "slower" more intense and sickening pain. The first one lasts very briefly and the second one is prolonged even after removal of the stimulus. The two pains are transmitted up two different sets of fibres. The touch receptors related to the hairs respond to slight movement of the hairs and adapt quickly but there are others which go on discharging for several minutes, as long as the mechanical stimulus usually lasts. These could be the ones concerned in signalling the feel of objects. Some other touch receptors appear to be concerned with a high degree of tactile localization, conveying this information up thicker myelinated fibres (Starling and Lovatt Evans, *ibid*).

It is well to remind ourselves that the sensitivity of the skin is punctate, with definite "spots" of high sensitivity to the four chief modalities and there are more sensitive pain spots than touch, much fewer cold, and still less warmth ones. A few interesting comparisons can be noted.

The highest density of pain spots per square centimetres are in areas which are not normally exposed to so many tactile stimuli, the thigh, the forearm, the breast, the back of the hand and the forehead, only half as many on the volar aspect of the fingers, the nose and the back of the fingers. Touch spots are particularly numerous on finger tips, tongue, then nose, and fewer on thigh and forearm and dorsal aspects. Cold spots are more

numerous around the mouth (Starling and Lovatt Evans, *ibid*).

The joint receptors also vary slightly in their receptivity to different deformations. Some of them with smaller myelinated fibres are stretch receptors signalling small movements. They are slowly adapting and discharge readily from the capsule and ligaments in different angles of movement. They are sensitive to the direction and speed of small movements. Other pacinian-like receptors have larger myelinated fibres and adapt rapidly and are very sensitive to rapid movements, they are also in the capsule.

Still larger receptors and fibres adapt very slowly and have a high threshold. They record joint position and are in the ligaments only. Lastly of course there are the fine pain nerve endings and the fine sympathetic fibre endings in the joints.

These receptors and their fibre types have been described in some detail in order to stress that there are a variety of sensory impulses being transmitted from two areas which we are treating repeatedly, the joints and the skin, and that they pass up large and small fibre systems. For those of us who are less familiar with it, a classification in summary of these modalities according to the diameter of the fibres in the first neurone is shown in Table 1.

It will be seen that the unpleasant slow pain fibres are the finest ones. Experimental work on humans proved conclusively that the burning, sickening, slow pain occurs when characteristic potentials for the C fibres are recorded on stimulation of a mixed nerve (Collins *et al.*, 1960).

It is a fact that conduction down the different groups of fibres can be slowed or blocked selectively in different ways and this may explain the way in which some physical agents act on pain. Sustained cold tends to inhibit transmission down the finer fibres. Anoxia

TABLE 1

Fibre Type	FUNCTION		*Diameter* μ	*Conduction Velocity Meters Per Second*
	Motor	*Sensory*		
Myelinated Group A				
α Alpha	Motor to extrafusal muscle	Primary stretch in Spindle Golgi tendon organ	12-20	70-120
β Beta		Secondary stretch in Spindle Fast touch — pressure — Joint position and movement sense	5-12	30- 70
γ Gamma	Motor to spindle intrafusal muscle			
δ Delta		Quick pain — Temperature Light touch	2- 5	12- 30
Group B	Pre-ganglionic Autonomic		< 3	3- 15
Non-myelinated or poorly myelinated Group C D.r.		Slow pain — Temperature Touch —	0.4-1.2	.5-2.0
Symp	Post ganglionic autonomic		.3-1.3	.7-2.3

Classification of Nerve Fibres

According to Erlanger and Gasser

affects the thicker fibres first and considerably later the smaller ones. We can relate this to what we know of the effectiveness of sustained cold in relief of pain. Pressure on the larger fibres from local swelling or other mechanical factors may well aggravate pain by cutting out stimuli up the larger fibres and facilitating the fine C fibres.

On the one hand there seems to have been considerable clinical evidence to support the fact that unpleasant painful sensations are associated with loss of conduction in some of the afferent fibres; the causalgia's related to partial nerve lesions, post herpetic pains (apparently the larger peripheral nerve fibres are lost in these cases (Blackwood *et al.*, 1963), paroxisms of spontaneous pain from stumps and hyperalgesic areas, all these often occur after only gentle stimuli and with considerable delays, suggesting conduction in a slow fibre system with a tendency for spontaneous discharges. (These are characteristic of C fibres).

Absence of small fibres in the dorsal roots of a patient who had congenital absence of pain was reported by Swanson, Buchan and Alvord in 1965.

On the other hand various medical authors have reported success in controlling pains of this nature with stimuli affecting afferent input (Bradley, 1955; Ellis, 1961; Taverner, 1961) and this many of us have confirmed over and over again. One wonders how far restoration of normal afferent stimuli from joints which are mobilized may help to control joint pain.

In their article "Pain Mechanisms, a New Theory" Ronald Melzack and Patrick Wall suggested a very interesting theory on the way in which lack of balance between small and larger sensory fibre transmission might be magnifying pain impulses through the influence of these fibres on the cells of the Substantia Gelatinosa Rolandi which are interposed between the first and second neurones in the dorsal horn. These cells exist throughout the length of the cord and intercommunicate at all levels. Melzak and Wall's theory has become known as the "Gate theory", illustrating a likely mechanism of pre-synaptic inhibition and is taught in Physiology Departments. (*See* Diagrams 1 and 2.)

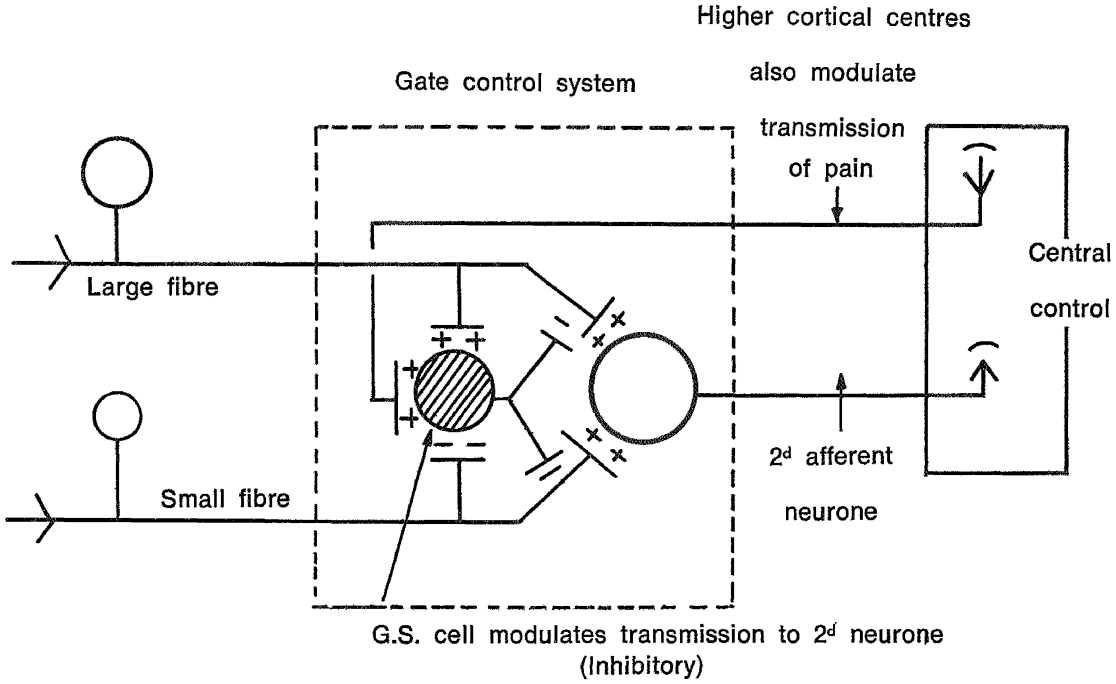

DIAGRAM 1

The mechanism of the Gate Control theory from Melzack and Wall.

Transmission at the second neurone is apparently also influenced from above (Brodal, *ibid*). Melzack and Wall's theory has been applied by Shealy (1967).

We know of course that selection of input must occur at many levels of the central nervous system, that the reticular formation and the cortex itself can act to facilitate or inhibit afferent messages.

However some neurophysiologists believe that many of our treatments could well be acting mainly on larger fibres and inhibiting pain at the spinal level through pre-synaptic inhibitory mechanisms. It is possible also that the duration and frequency of certain impulses are related to their degree of success as inhibitors. Low frequency currents in sinusoidal form and around 100 cycles per second may act on the larger sensory fibres. They are effective pain inhibitors (Sinusoidal and Interferential).

Opportunity for frequent communication with the patient, treatment of different types of pain with many different physical agents, constant observation of results should place the physiotherapist in a good position to be able to assist the anatomist and the physiologist who are gradually trying to unravel the intricacies of the anatomy of pain pathways and the physiology of pain mechanisms and the physician who is making the diagnosis and prescribing treatment but also welcoming suggestions for effective treatment. It is interesting to find that research workers in this field not infrequently use isolated clinical examples to substantiate some of their theories, quoting the effectiveness of a particular afferent stimulus to relieve pain when these stimuli have already been used for years, repeatedly and successfully by physiotherapists.

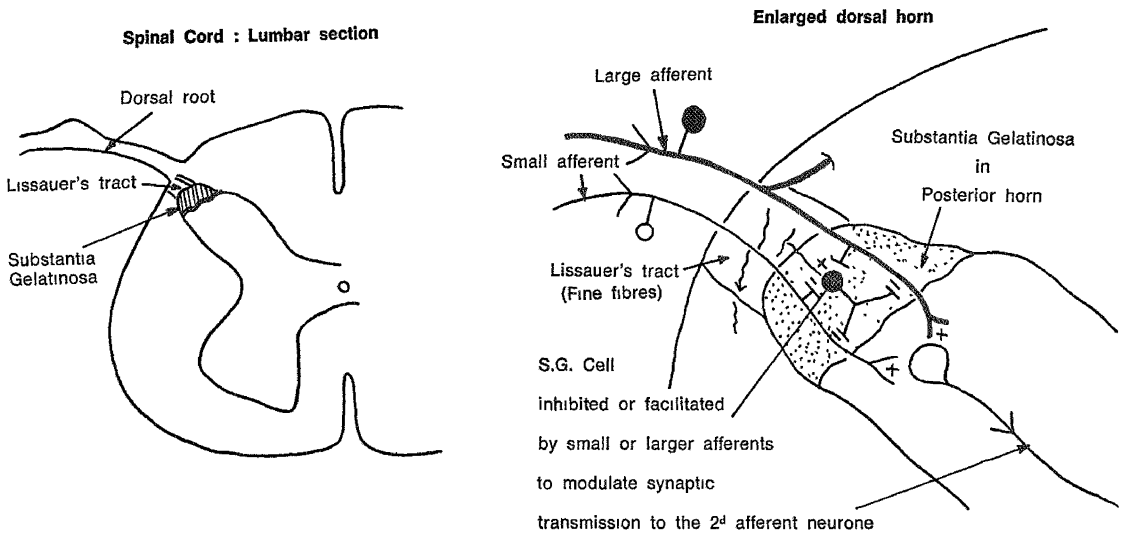

DIAGRAM 2

Meanwhile some members of the medical profession, and others in our own profession belittle the value of physical agents through ignorance and lack of experience. It is really unnecessary to waste time on this attitude but let us find out more with the help of Departments of Physics and Physiology before others do so and claim the work as their own because they have the time and the finance to do so. We are not sufficiently curious and, of course, time is always lacking to allow us to keep up-to-date with recent physiological knowledge that could stimulate us to direct a little more of our time to "why".

SUMMARY

The importance of restoration of function in helping to prevent recurrence of painful states has been suggested, particularly in relation to re-establishment of normal afferent input.

A review of some of the present knowledge on afferent receptors and fibres from the skin and joints has been given, with some of the theories on local pain mechanisms.

An attempt has been made to correlate some clinical findings with knowledge of the possible effects of physical agents on the control and abolition of pain.

Melzack and Wall's theory on the need for a balance between small and large fibre inputs has been recalled and the part that the physiotherapist can play is stressed.

REFERENCES

BLACKWOOD, W. (1963), *Greenfield's Neuropathology*, Arnold, London.

BRADLEY, KEITH, F.R.A.C.S. (1955), *"The Sequelae of Amputation"*. Research undertaken for the Services Canteen Trust.

BRODAL, A., M.D. (1969), *"Neurological Anatomy in Relation to Clinical Medicine"*, 2nd edition, Oxford University Press, New York, London, Toronto.

COLLINS, W. F. JR., NULSEN, F. E., and RANDT, C. T. (1960), "Relation of Peripheral Nerve Fibre Size and Sensation in Man", *Arch. Neurol. (Chic.)* 3: 381-385.

ELLIS, M., M.A., M.B., CHIR, B., F.R.C.S. (1961), "The Relief of Pain by Cooling of the Skin", B.M.J. (i), 250-252.

GERTSEN, J. W. (1955), "Effect of Ultra Sound on Tendon Extensibility", *Am. J. of Phys Med.*, April, 34:362.

LIM, ROBERT K. S. (1970), "Pain", *Annual Review of Physiology*, 32: 269-284.

MELZACK, R., and WALL, P. (1965), "Pain Mechanisms, a new theory". *Science*, 150, 2 : 971-979.

SHEALY, M.D., et al. (1967), "Electrical Inhibition of Pain: experimental evaluation", *Anesthesia and Analgesia*, 46, 3: 299-304.

"Current Status of Dorsal Column Stimulation for Relief of Pain".

Proceedings of the International Headache Symposium, Elsinore, Denmark, May 16th, 18th, 1971.

STARLING and LOVATT EVANS (1968), *"Principles of Human Physiology"*, 14th edition. Edited by Hugh Davson, D.Sc. and M. Grace Eggleton, D.Sc., M.R.C.S., J. A. Churchill Limited, London.

SWANSON, A. G., BUCHAM, ALFORD (1965), Archives of Neurology, 12, 12.

TAVERNER, D., M.B.E., M.D., LEEDS, F.R.C.P. (1960), "Alleviation of Post Herpetic Neuralgia", *Lancet ii* : 671.