Neuropathic Pain Unveiled (NPU) Model for Patient Education

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ABSTRACT
Neuropathic pain (NP) is defined as pain initiated or caused by a primary lesion or dysfunction of the nervous system. It is one of the most challenging conditions with respect to understanding the complex relationship between symptoms, mechanisms and various approaches to treatment. Recent estimates suggest that up to 8% of the general population suffers with pain associated with neuropathic features and when it persists, it impacts significantly on patients’ lives affecting physical, physiological and social functioning. Neuropathic Pain Unveiled (NPU) is a novel portable visual-electronic model which can educate patients, pain nurses and medical students about the complex mechanisms of NP and help them understand the mode of action of various pharmacological agents used in the treatment of NP.

Keywords
Neuropathic pain, ATMEL 89C51.

1. OBJECTIVE
NP is a chronic pain condition where pain persists for longer than 3 months. This is not only devastating for patients but also places considerable demands on society including financial burdens relating to health care costs, disability and benefits.

Education about the pain is a key component of any pain management programme as inability to comprehend the reason for their pain is very frustrating to patients and can hinder successful pain management strategies. Our NPU model can explain and educate about the complex mechanism of neuropathic pain. [8,9].

2. CENTRAL NERVOUS SYSTEM AND PERIPHERAL NERVOUS SYSTEM
It is the control and communication network for the body which coordinates the functions of various organs. Rapid communication between various parts, effective, integrated activity of different organs and tissues and coordinated contraction of muscle are almost entirely dependent upon the nervous system. It is thus, most highly developed and complex system in the body.

The nervous system is the body’s principal regulatory system and pathological processes in it often lead to serious functional disturbances (like NP). The symptoms vary greatly depending upon the part of nervous system affected by pathological changes [3].

The central nervous system (CNS) is the part of the nervous system that integrates the information that it receives from, and coordinates the activity of all multicellular animals except sponges and radially symmetric animals such as jellyfish. It contains the majority of the nervous system and consists of the brain and the spinal cord. Together with the peripheral nervous system, it has a fundamental role in the control of behaviour. The peripheral nervous system, or PNS, consists of the nerves and ganglia outside of the brain and spinal cord. The main function of the PNS is to connect the central nervous system (CNS) to the limbs and organs. The peripheral nervous system is divided into the somatic nervous system and the autonomic nervous system; some textbooks also include sensory systems [1,2].
2. ATTRIBUTES OF NEUROPATHIC PAIN
Abnormal and peculiar nature of NP [5,6] has various features:
1. Spontaneous pain that is continuous in nature includes unpleasant or abnormal sensation felt in the skin (dysaesthesia) described as burning, tingling, itching or pins & needles.
2. Deeper pain may be described as aching, cramping or crushing.
3. Paroxysmal element is often described as shooting, stabbing or electric-shock like pain.
4. Allodynia describes pain that is experienced from a stimulus which would normally go unnoticed such as skin contact with clothing or cold breeze.
5. Hyperalgesia which is an exaggerated painful sensation after a painful stimulus.

3. PERIPHERAL AND CENTRAL MECHANISMS
Mechanisms postulated at peripheral and central (brain and spinal cord) levels [7] are:
1. Increased sensitivity of damaged nerve to mechanical and chemical stimuli which will lead to hyperalgesia.
2. Ectopic discharge from the damaged nerve which will lead to spontaneous nature of this pain with abnormal sensation (dysaesthesia).
3. Anatomical re-organization or ephaptic cross connection/rewiring which can occur within the spinal cord where the nerve fibres normally involved in touch and pressure sensation make sprout connections in the area where pain fibres are occupied as a result previously non-painful stimuli may now be experienced as painful (Allodynia).
4. Cerebral Plasticity: Even after complete recovery from primary lesion for NP, these patients may still feel abnormal pain for long which is due to morphological changes in the brain and because of these patients may still feel non-painful stimuli as painful (Allodynia).
5. Inhibition of descending inhibitory tracts; the function of these tracts is to slow down or stop pain sensation transmission at spinal cord.
6. Activation of descending facilitatory tract resulting in increased pain sensation transmission at spinal cord.

5. AVAILABLE TOOLS AND REMEDIES
Several tools are available to distinguish nociceptive from neuropathic pain. Tools that combine self-report and physical examination are more precise than self-report alone. Validation of the following three tools has included some, but not large numbers, of older adults.

6. STRENGTHS AND LIMITATIONS
Although the three tools described distinguish nociceptive from neuropathic pain, the LANSS and DN4 are preferred because of their brevity and the integration of self-reported symptoms and physical examination. But still distinguishing pain types by linking signs, symptoms and responses remains an active area of research. Thus, the underlying mechanisms of pain are better understood, and targeted therapies a developed to minimize treatment failures and expedite relief provide with sound results.

7. NEUROPATHIC PAIN UNVEILED (NPU) MODEL
A Neuropathic Pain Unveiled (NPU) model in Fig.1 is a microcontroller based printed circuit board (PCB) which is sequentially programmed with 5 different colours multiple light emitting diodes. Three diagrammatic components include, body part, transverse section of spinal cord with laminae and coronal section of brain. LEDs form transmission tracks from body part to brain via spinal cord which on illumination not only explain the mechanism of feeling normal touch and nociceptive pain (normal pain) but also demonstrate abnormal nature of pain in NP condition (sympathetic dysfunction and occasional dystrophy) at different levels of transmission pathway. Essential parts are driver circuit, microcontroller development board and LEDs.

8. ATMEL 89C51 TECHNICAL DESCRIPTION
The AT89C51 [4], is an 8051-based Fully Static 24MHz CMOS controller with 32 I/O Lines, 2 Timers/Counters, 6 Interrupts/2 Priority Levels, UART, Three-Level Program Memory Lock, 4K Bytes Flash Memory, 18 Bytes On-chip RAM.
Fig.3 Various pain mechanisms and their remedies shown by coloured pathways

In our model it plays the most important part of sequence generation. The differently coloured LEDs derive their input voltages/current levels from the above mentioned microcontroller with respect to the specific code written. The code is written with the help of c language in programmer’s notepad and Win AVR is used to program the microcontroller. The code thereby monitors the specific and sequential firing of LEDs and also the pace of firing. According to the need of the model, the code written drives the LEDs in a predefined sequence to illustrate various pain mechanisms and their corresponding pathways are illuminated. For example: Red LEDs will illuminate from laminae I of spinal cord reaching up to sensory cortex of brain suggesting normal touch sensation.

9. DIAGRAM AND DESCRIPTION OF NPU MODEL

Model (Fig.1) has an external power supply and remote-control operated various functional features which include:

A. Touch Sensation: On activation will transmit signal and sequentially illuminate green LEDs from body part through laminae IV of spinal cord all the way up to sensory cortex of brain demonstrating normal touch sensation.

B. Nociceptive Pain: On activation will sequentially illuminate red LEDs from body part to laminae II of spinal cord and all the way up to sensory cortex of brain demonstrating normal pain sensation.

C. Neuropathic Pain (NP)

1) NP-1: On activation will sequentially illuminate green LEDs from body part to spinal cord laminae IV, but then activate orange LEDs which are clustered between laminae II and IV, further stimulating/activating pain pathway (red LEDs) all the way up to brain suggesting rewiring mechanism at spinal cord level for allodynia component of NP.

2) NP-2: On activation will stimulate pain transmitting tract by rewiring mechanism which is taking place at peripheral pathway (between body part and spinal cord).

3) NP-3: Random, spontaneous stimulation and activation of orange LEDs can be at peripheral pathway or between laminae II and IV of spinal cord which will further activate red LEDs suggesting pain transmission. This mechanism suggests ectopic pacemaker activity.

4) NP-4: On activation will increase the illuminating speed of red LEDs (pain transmission) from spinal cord to brain sensitization mechanism for hyperaesthesia component of NP.

5) DIT (Descending Inhibitory Tract): Decreasing transmission/illumination speed of blue LEDs from brain to laminae II of spinal cord, which will speed up the illumination of red LEDs from spinal cord to brain suggesting inhibition of descending inhibitory tracts.

6) DFT (Descending Facilitatory Tract): Increasing transmission/illumination speed of purple LEDs spinal cord laminae II, which will speed up the illumination of red LEDs from spinal cord to brain suggesting activation of descending facilitatory tract.

7) CP (Cerebral Plasticity): Scattered illumination of bright white LEDs followed by illumination of red LEDs in the brain only suggesting CP.

D. Treatment: Various and non-pharmacological treatments will act at different levels on this model which on activation will show following features:

1) Slow down/stop abnormal rewiring mediated transmission in orange LEDs.

2) Slow down/stop abnormal activation of red LEDs mediated transmission both peripherally as well as centrally.

3) Inhibit activation of purple LEDs and hence slow down/stop signal transmission by red LEDs from spinal cord to brain.

4) Dull down transmission in scattered white and red LEDs in the brain.

10. CONCLUSION

It is proposed that NPU model will be a very significant advancement in the field of instrumentation and medical electronics. Doctors may convince the patients and their relatives very effectively using such a designed visual presentation, for communication in verbal or through printed words has its own limitations but when it is through visuals it becomes all the more easy to comprehend and get convinced. The primary goal of our paper is to identify action initiatives related to the crucial aspects of Neuropathic Pain and its diagnosis techniques. The central idea lies in fostering such an inclusive and responsive environment for neuropathic patients where all the patients are valued equally and treated with respect. Our model thus helps them to understand the basis of various treatment options; in turn helping them cope better with their pain.

NPU model will inevitably evolve as a useful tool in the hands of concerned medical practitioners.
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12. REFERENCES


