EAU Guidelines on Pain Management

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Abstract

Pain is the most common symptom of any illness; the physician’s therapeutic task is twofold: to discover and treat the cause of pain and the pain itself, whether or not the underlying cause is treatable, to provide relief and reduce the suffering caused by pain. Although we use the term of pain to define all sensations that hurt or are unpleasant, actually two quite different kinds of pain exist. The first (nociceptive) is associated with tissue damage or inflammation, the second (neuropathic) results from a lesion to the peripheral or central nervous systems. Pain can also be divided in acute and chronic.

Caregivers are to face pain in two main settings: after surgery and in cancer patients. These tasks require a multidisciplinary team, able to properly assess and treat pain. Postoperative pain is to be treated early and aggressively. Several drug options are available, to be tailored on the surgical procedure and the patient. Pain in cancer patients consists of different aspects: it can be caused by the cancer itself or may be secondary to muscular spasm or cancer treatments. The management involves mainly pharmacotherapy, but also primary treatments as surgery, radiochemotherapy or even antibiotics can provide an adequate relief.

Analgesics are to be employed according to an ascending scale, but other options can be combined to improve the outcome when a satisfactory balance between relief and side effects is not achieved; they include invasive techniques, physical and psychological therapy.

The mainstay of pain management entails a interdisciplinary cooperation; it requires a full knowledge of the methods of evaluation and treatment of this condition.

Keywords: Pain management; Surgery; Urogenital neoplasms

1. Definition of pain

According to the International Association for the Study of Pain (IASP) pain is “an unpleasant sensory and emotional experience associated with either actual or potential tissue damage, or described in terms of such damage” [1]. Two different kinds of pain exist. The first is termed nociceptive because of its direct link with noxious stimuli: it is a key component of the body’s defence mechanisms and is associated with tissue damage or inflammation, so it is also called inflammatory pain. The second is defined neuropathic pain and results from a lesion to the peripheral or central nervous systems. Pain can also be divided in acute and chronic. Acute pain arises after trauma, surgery, or nerve damage. Chronic pain persists beyond 3 months: it inhibits feelings and emotions, thought and reactions, and may restrict social interactions and work.

2. Nociception and innervation

The sensation of pain starts at the site of tissue injury. The peripheral neural apparatus alerts the organism of potential injury sending messages to the central nervous system regarding the location and intensity of noxious stimuli. It operates through highly specialized sensory
fibres and a specific set of primary sensory neurones called “nociceptors”, subclassified as unmyelinated (C-fibre) versus myelinated (A-fibre) parent nerve fibre. Unlike cutaneous pain, deep pain is diffuse and poorly localized. It may be associated with strong autonomic responses such as sweating and changes in heart rate, blood pressure and respiration. In addition, deep pain may be produced also by not-tissue-damaging stimuli, e.g. distension of bowel and bladder [2].

The nociceptors terminate in a highly ordered way in the dorsal horn of the spinal cord; the unmyelinated C-fibres activate a large number of second order interneurones in the spinal cord. The activity is transferred directly, or via brainstem relay nuclei, to the thalamus and then up to the cortex, where the sensation of pain is generated. Numerous brain areas are involved in the various components of pain: the sensation of pain is subject to modulation during its ascending transmission from the periphery to the cortex but also to segmental modulation and descending control from higher centres. The main neurotransmitters implicated in descending control are serotonin, noradrenaline and endogenous opioids.

3. Innervation of the urogenital system

3.1. Ureter

Ureteric afferents are thinly myelinated or unmyelinated and two populations of fibres can be distinguished [3]. The first responds to contractions of the ureter and can be excited by low levels of distension. The second group can be excited by distension with a wide range of thresholds.

3.2. Bladder

Two groups of afferent fibres carry noxious stimuli: most visceral afferents are unmyelinated fibres, while a subpopulation of C-fibres is excited by chemical irritants, thus confirming the role of mucosal inflammation in intensifying pain [4]. Nearly all afferents travel with sympathetic (hypogastric) or parasympathetic (pelvic) nerves. Distension excites mainly the thin myelinated afferents, with pressure thresholds corresponding to the values where humans report the first sensation of fullness. There are only a few specific nociceptors in the bladder, capable to signal only painful levels of distension at an intravesical pressure of 50 mmHg.

3.3. Male reproductive organs

Free nerve endings, derived from Ad- or C-fibres are abundant throughout the glans penis, and the associated fibres appear to be slowly adapting low-threshold stretch receptors and high-threshold mechanoreceptors [5]. Either the fibres of the superior spermatic nerve (mainly unmyelinated) or those from testis and epididymis present polymodal properties (i.e. responding to mechanical, chemical and thermal stimuli).

4. Postoperative pain management

4.1. Importance of postoperative pain control

Postoperative pain is expected by 77% of adults and represents the primary fear for nearly 60% of them before surgery [6]. Surgical trauma induces the release of potent mediators of inflammation and pain, capable to evoke stress hormone responses in addition to activation of cytokines, adhesion molecules and coagulation factors. This response leads to an increase in metabolic rate and water retention and, finally, to pain and surgical morbidity. The common practice of giving “as required” intramuscular opioids does not relieve pain in over 50% of patients [7] (E.L. III). Unrelieved pain can lead to many adverse effects, both cardiovascular and respiratory.

4.2. Methods in treating postoperative pain

4.2.1. Development of acute pain teams

In order to oversee effective postoperative pain control, back in the 1980s multidisciplinary teams were developed, lead by an anesthesiologist, consisting of nursing and pharmacy personnel. Pain relief is obtained by means of regular pain assessment, easy access to strong opioid drugs, teaching and education. These “low tech low cost” approaches appear to be as important as Patient Controlled Analgesia (PCA) and epidurals, thus potentially leading to shorter hospital stays [8] (E.L. III).

4.2.2. Preoperative interventions

Postoperative pain management should be discussed by the surgeon and the anaesthesiologist before the planned operation, including an assessment of preoperative pain, previously used analgesic methods, patient’s preferences for pain management. A pain measurement tool should be selected (e.g. Visual Analogue Scale or Descriptive Scale) and the patient told how often pain will be assessed: self-reporting techniques are useful in obtain a regular assessment throughout treatment.

4.2.3. Postoperative management

It is considered good clinical practice to treat postoperative pain early and aggressively (“preventative” analgesia [9], E.L. IIIb).
### Table 1
**Analgesic drugs**

<table>
<thead>
<tr>
<th>Class</th>
<th>Drug</th>
<th>Dosage</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSAIDs</td>
<td>Diclofenac</td>
<td>50 mg; 3× per day</td>
<td>orally</td>
</tr>
<tr>
<td></td>
<td></td>
<td>100 mg; every 16 hours</td>
<td>rectally</td>
</tr>
<tr>
<td></td>
<td>Ibuprofen</td>
<td>200–400 mg; 3×</td>
<td>orally</td>
</tr>
<tr>
<td></td>
<td>Ketorolac</td>
<td>10–30 mg; 6–4×</td>
<td>orally or IV</td>
</tr>
<tr>
<td></td>
<td>Rofecoxib</td>
<td>20 mg; 1×</td>
<td>orally</td>
</tr>
<tr>
<td>COX inhibitors</td>
<td>Paracetamol</td>
<td>1 g; 4×</td>
<td>orally or rectally</td>
</tr>
<tr>
<td></td>
<td>Co-codamel</td>
<td>2 tab; 4×</td>
<td>orally</td>
</tr>
<tr>
<td></td>
<td>Co-proxamol</td>
<td>2 tab; 4×</td>
<td>orally</td>
</tr>
<tr>
<td>Opioids</td>
<td>Tramadol</td>
<td>50–100 mg; 4× or cont.</td>
<td>orally or IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Loading dose 100 mg + 0.2 mg/kg (maintenance)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Morphine</td>
<td>5–10 mg; 8–6×</td>
<td>orally</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Up to 10 mg per hour</td>
<td>IV/SC infusions</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10 mg; 8×</td>
<td>IM/SC injections</td>
</tr>
<tr>
<td></td>
<td>Oxycodone</td>
<td>10 mg; 6×</td>
<td>orally</td>
</tr>
</tbody>
</table>

*a* Codeine + Paracetamol 500 mg.

*b* Dextropropoxyphene 32.5 mg + Paracetamol 325 mg.

### Table 2
**Analgesic techniques**

<table>
<thead>
<tr>
<th>Technique</th>
<th>Drug(s)</th>
<th>Schedule</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCA&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Morphine 1–2 mg</td>
<td>Loading dose: 0.05–0.2 mg/kg</td>
</tr>
<tr>
<td></td>
<td>Pethidine 10 mg</td>
<td>Incremental dose: M-P-F</td>
</tr>
<tr>
<td></td>
<td>Fentanyl 20 mcg</td>
<td>Lockout period: 5–8 minutes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Background infusions (close monitoring)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 hour infusion limit</td>
</tr>
<tr>
<td>Epidurals</td>
<td>Bupivacaine 0.125%</td>
<td>+ fentanyl 2 mcg/ml (or sufentanyl 0.05–0.1 mcg/ml), run at 5–15 ml/h</td>
</tr>
<tr>
<td></td>
<td>Ropivacaine 0.1–0.2%</td>
<td></td>
</tr>
<tr>
<td>Nerve blockade</td>
<td>Bupivacaine 0.25–0.5%</td>
<td>Wound or iliohypogastric/ilioinguinal nerve infiltration (10–20 ml)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intercostal nerve infiltration (5–10 ml)</td>
</tr>
<tr>
<td></td>
<td>0.25%</td>
<td>Intrapleural catheters, continuous infusion (10 ml/h)</td>
</tr>
<tr>
<td></td>
<td>0.25%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.1%</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> Patient Controlled Analgesia.

### Table 3
**Analgesic drug options after urological procedures**

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Drug</th>
<th>Route</th>
<th>Schedule</th>
</tr>
</thead>
<tbody>
<tr>
<td>ESWL</td>
<td>Diclofenac</td>
<td>orally</td>
<td>8 h</td>
</tr>
<tr>
<td>Transurethral surgery</td>
<td>Paracetamol 1 g</td>
<td>orally</td>
<td>6 h</td>
</tr>
<tr>
<td>Percutaneous surgery</td>
<td>Co-proxamol; Co-dydramol</td>
<td>2 tab.</td>
<td>6 h</td>
</tr>
<tr>
<td>Minor operations</td>
<td>Tramadol 50–100 mg</td>
<td>orally</td>
<td>6 h</td>
</tr>
<tr>
<td>Transvaginal surgery</td>
<td>Morphine 10 mg</td>
<td>IM</td>
<td>3 h</td>
</tr>
<tr>
<td>Laparoscopic surgery</td>
<td>as above plus:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>PCA morphine 1 mg</td>
<td>bolus</td>
<td>5 min lockout</td>
</tr>
<tr>
<td>Perineal open surgery</td>
<td>as laparoscopic surgery plus:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Laparotomies (all) Flank incisions</td>
<td>Bupivacaine 0.25% + Fentanyl 2 mcg/ml</td>
<td>epidural infusion</td>
<td>5–15 ml/h</td>
</tr>
<tr>
<td></td>
<td>Morphine</td>
<td>IV</td>
<td>1–10 mg/h</td>
</tr>
<tr>
<td></td>
<td>(+ bolus doses 1–2 mg as required)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Commonly used analgesic drugs include nonsteroidal anti-inflammatory drugs (NSAIDs), COX inhibitors, opioids and metamizole [10] (Table 1). Alternatives are represented by Patient Controlled Analgesia [11] (PCA: the patient operates a machine which results in drug delivery directly in the blood stream), and continuous epidural infusions, able to provide superior analgesia compared to PCA or i.m. opioid [12]. Moreover, local anaesthetic blocks can be used to supplement postoperative analgesia (Table 2).

### 4.3. Specific pain treatment after surgery

Table 3 outlines a recommended postoperative approach to most common urological procedures.

### 5. Treatment of cancer pain

Pain in patients with cancer is a complex phenomenon consisting of many different aspects. Not all pains will be of malignant origin: a key principle is to constantly re-evaluate pain and the effect and side-effects of analgesic therapy. Pain may be caused by the cancer itself (e.g. tumour pressure on nerve plexus or tumour infiltration) or may be due to secondary muscular spasm. Additionally, it may be secondary to cancer treatments, e.g. radiation induced brachial plexopathy, or may have no relation to the cancer, e.g. arthritis.

In general cancer pain consists of two broad diagnostic types, nociceptive and neuropathic pain. Nociceptive pain includes bone pain and soft tissue pain; typically, it is described as a dull, well localised and aching pain, largely sensitive to non-steroidal anti-inflammatory drugs and opioids. Neuropathic pain results from damage to the peripheral or central nervous system. Not particularly responsive to NSAIDs or opioids, it is usually described as a burning or sharp, shooting pain. Adjuvant analgesics such as anti-depressants and anti-convulsants should be used in the first instance.

Urogenital neoplasms frequently metastatise to bone (e.g. spine, pelvis, skull) and such bone metastases are associated with pathological fractures, hypercalcaemia and neurological deficits, which may result in pain and impairment of quality of life. The release of pain-provoking substances in the tissue, microfractures and periostial tension are the main mechanisms for pain sensation [13]. Pain caused by bony metastases is nociceptive pain, but can become associated with neuropathic pain if the tumour invades or compresses a nerve, neural plexus or spinal cord. A third of patients with tumour-related pain are affected by neuropathic pain components [14]. The efficacy of opioids may be diminished in neuropathic pain and hence additional co-analgesics are necessary.

#### 5.1. Pain measurement

Pain is a multidimensional complex phenomenon and is not adequately described by unidimensional scales, although a number of different rating scales have been devised to attempt to methodically measure it:

- Categorical scales, e.g. verbal rating scales, mild, moderate, severe pain;
- Visual analogue scale (VAS), e.g. a line is drawn with numbers from 0 (no pain) to 10 (severe pain), pain severity is indicated by marking along the line;
- Complex pain assessment compendiums, e.g. Brief Pain Inventory (BPI), consisting of several visual analogue scales grouped together assessing pain at rest on movement and other aspects of the pain including interference with function and effect on work.

#### 5.2. Management

General principles include:

- Treatment individualisation: in some cases surgical measures such as drainage and stenting can make analgesic medication redundant.
- Anti-cancer therapies should be used first (e.g. surgery, chemotherapy, radiotherapy).
- Use analgesic drugs according to WHO ladder (Fig. 1).
- Utilise both psychological counselling and physical therapy throughout.

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**Fig. 1. The ‘analgesic ladder’ according to WHO.**
5.2.1. Primary analgesic therapies

- **Radiotherapy** has a major role in the treatment of cancer pain, mainly of bone metastases [15].
- **Chemotherapy** has a successful effect on pain, generally depends on the tumour response.
- **Surgery** may have a role in the relief of symptoms caused by specific problems, such as obstruction of a hollow viscus, unstable bony structures and compression of neural tissues.
- **Antibiotics** may be analgesic when the source of the pain involves infection (e.g. pyonephrosis, abscess, and osteitis pubis).

5.2.2. Pharmacotherapy

Systemic analgesic pharmacotherapy is the mainstay of cancer pain management. Analgesic drugs can be separated into three groups:

1. nonopioid analgesics
2. opioid analgesics
3. adjuvant analgesics, which are drugs with other primary indications that can be effective analgesics in specific circumstances.

The Cancer Unit of the World Health Organization (WHO) has proposed a three-step approach to drug selection for cancer pain, which has become known as the ‘analgesic ladder’ (Fig. 1) [16].

**Step 1:** Mild to moderate cancer-related pain should be treated with a nonopioid analgesic, combined with an adjuvant analgesic if required.

**Step 2:** Moderate to severe pain, or which fails to achieve adequate relief after a trial of a nonopioid analgesic, should be treated with a weak opioid. This treatment typically entails a combination of a nonopioid (e.g. aspirin or acetaminophen) and an opioid drug (such as codeine, oxycodone or propoxyphene). They can also be coadministered with an adjuvant analgesic.

**Step 3:** A strong opioid, such as morphine or hydromorphine (+/- combined with an adjuvant drug), should be administered for severe pain, or when adequate relief following appropriate administration of drugs on the second rung of the ‘analgesic ladder’ is not obtained.

**5.2.2.1. Nonopioid analgesics.** Nonopioid analgesics include aspirin, acetaminophen and NSAIDs. They may be useful alone for mild to moderate pain (Step 1 of the analgesic ladder), provide analgesia when combined with opioids and entail a *ceiling effect* of analgesic efficacy but no tolerance or physical dependence. Mechanism of action involves the inhibition of the enzyme cyclo-oxygenase blocking the synthesis of prostaglandins. Most common adverse effects [17] include bleeding diathesis due to inhibition of platelet aggregation, gastro-duodenopathy and renal impairment.

**5.2.2.2. Opioid analgesics.** Based on their interactions with the various receptor subtypes, two subtypes can be distinguished:

- **agonist:** most commonly used in clinical pain management, with no ceiling effect for analgesia;
- **agonist-antagonist:** with ceiling effect.

Whilst patients with moderate pain are commonly treated with a combination drug containing acetaminophen or aspirin plus codeine, oxycodone or propoxyphene, severe cancer pain should generally be treated with a systemically administered, ‘strong’ opioid from the start.

Opioid selection requires some factors are considered: pain intensity, patient age, prior opioid therapy, coexisting diseases and influence of underlying illness. They should be administered by the least invasive and safest route capable of providing adequate analgesia.

The oral route of administration is the preferred approach in routine practice but, when not available, can be replaced by the parenteral route. Intravenous bolus administration provides the most rapid onset and shortest duration of action: time to peak effect ranges from 2–5 minutes for methadone to 10–15 minutes for morphine. The switch between oral and parenteral routes should be guided by individual titration of dosage with adequate monitoring. If pain breaks through the regular schedule, a supplemental (rescue) dose can be offered on an “as needed” basis, also beneficial when rapid dose escalation is needed or therapy is begun with a long half-life opioid such as methadone or levorphanol. An alternative is “Patient Controlled Analgesia” (PCA), in which the patient controls an infusion device that delivers subcutaneously a bolus of analgesic drug ‘on demand’ according to parameters set by the physician.

Adverse effects include constipation (most common), respiratory depression (most serious), nausea and vomiting (mainly at the start of treatment), tolerance and addiction.

**5.2.2.3. Adjuvant analgesics.** Defined as drugs with a primary indication other than pain but analgesic in some conditions, these drugs may be combined with primary analgesics in any of the three steps of the ‘analgesic ladder’ to improve the outcome for patients who cannot otherwise attain an acceptable balance between relief and side-effects. They are classified in three groups:
A. multipurpose adjuvant analgesics:
- corticosteroids: with antioedema and anti-inflammatory effect,
- neuroleptics: to associate anxiolytic and antiemetic effects,
- benzodiazepines: to treat pain-associated anxiety and insomnia;

B. adjuvants for neuropathic pain:
- tertiary and secondary amine tricyclic antidepressants,
- Gabapentin,
- clonidine: used transdermally in opioid-refractory patients;

C. adjuvants for bone pain:
- NSAIDs and corticosteroids,
- bisphosphonates (mainly Pamidronate),
- radiopharmaceuticals (Strontium$^{89}$, Samarium$^{159}$, Rhenium$^{186}$).

5.2.3. Other techniques
Ten to 30% of patients with cancer pain do not achieve a satisfactory balance between relief and side effects using systemic pharmacotherapy alone without unacceptable drug toxicity [16]. Therefore, other options are worth considering:

1. Transcutaneous Electrical Nerve Stimulation (TENS), used for mild to moderate musculoskeletal or neuropathic pain [18].
2. Invasive techniques. They include epidural, intrathecal and intraventricular opioid application, chemical rhizotomy, cordotomy and pituitary ablation.
3. Physical/psychological therapy. Electrical stimulation, heat or cryotherapy, pneumatic pump or orthotic devices can optimise some functions in chronic pain patients. Cognitive behavioural interventions and relaxation methods help decrease the perception of distress and muscular tension engendered by pain [19].

6. Pain management in urological malignancies

Pain may be directly attributable to tumour infiltration in three main areas: bone, nerve or a hollow viscus. Treatment entails many responses, including surgery, radiochemotherapy and pharmacotherapy.

6.1. Prostate cancer
Pain can occur in both early and advanced stages: it could be caused directly by cancer (77%), related to the cancer treatment (19%) or unrelated to either (3%) [20]. The overall incidence of chronic pain in prostate cancer patients is about 30–50%, but as patients enter the terminal phase of their illness this figure rises to 90% [21]. Surgery can successfully manage bladder outlet or ureteric obstruction (TURP, nephrostomy), and mechanical ileus due to infiltration of the rectum. Hormone and radiation therapy are useful in relief of pain due to bone metastases.

6.2. Transitional cell carcinoma
In bladder carcinoma pain can be present during the natural history of the disease, early as a burning pain together with irritative symptoms, or late in the advanced disease due to local invasion of neighbouring tissues or metastatic organ invasion. In TCC of the upper urinary tract pain is an initial symptom in around 30% of the cases [22]. Endoscopic (TURBT, nephrostomy) or open surgery (cystectomy, nephroureterectomy, bowel excision) are effective in many cases, while radiotherapy has a palliative analgesic role both in pelvic progression and bone metastases.

6.3. Renal cell carcinoma
Renal cell carcinoma is mainly diagnosed incidentally. Pain cannot be expected unless a tumour invades surrounding areas or obstructs the outflow of urine owing to haemorrhage and subsequent formation of blood clots. Surgery is the only effective management of this type of tumour, in terms of bleeding control, pain (even due to bone metastases) or paraneoplastic syndromes. In bone metastases radionuclide therapy with Strontium$^{89}$ seems to achieve good pain relief [23], while immunotherapy and hormonal or chemotherapy have no proven benefit in pain relief in this setting. Immuno-chemotherapy (IL-2 + IFN + 5FU) is highly effective in soft tissue metastases [24,25].

6.4. Adrenal tumours
They are rare. Metastatic carcinomas have a poor prognosis (mean survival: 4 months), so there is no literature concerning chemo- or radiotherapy and pain relief. Pain arising from soft tissue or bone metastases by malignant pheochromocitoma can be treated with $^{131}$J-MIBG, if the tumour takes up this radionuclide; radiation can be useful in adrenocortical carcinomas [26].

6.5. Penile cancer
Pain does not develop in proportion to the extent of the local tumour and usually is not a presenting complaint [27]. It can occur in both early and advanced stages of penile cancer, caused by local pressure from the tumour mass or infiltration of hollow viscous organs and by lymphoedema of the scrotum and lower limbs,
to be treated with physiatric techniques. In early stages, acute pain could be the result of a voiding dysfunction (subvesical obstruction). In advanced stages of the disease, it is usually caused by metastases or lymph node involvement; chemotherapy plus surgery or radiotherapy can play a role in this setting.

6.6. Testicular cancer

Approximately 20% of patients present with scrotal or inguinal pain, which disappears after orchietomy. Only 11% of the patients complain back or flank pain on initial presentation. Retroperitoneal lymph node metastases can cause hydronephrosis: pain can be relieved by ureteral stenting or nephrostomy insertion. Primary advanced tumour with pain due to bone metastases is very rare, maximal no more than 3% at first presentation [28] and should be treated causally by primary chemotherapy and adjuvant analgesics.

7. Conclusions

Effective relief of pain should utilise a multidisciplinary, comprehensive approach. Those concerned with the care of pain must have adequate knowledge of all the therapies to employ. Side effects caused by the inappropriate use of anti-cancer treatments can be very distressing, and in all cases the disadvantages of a treatment must be balanced against the palliative benefit. Thus, the best approach to pain relief will be through interdisciplinary cooperation.

References


