Optimum VA

NAVAO Newsletter Spring 2007

News and Notes

99202

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President's Report

Gay Tokumaru, OD

As was announced on the March 16 conference call, there will be some changes to the NAVAO annual banquet starting this year. Starting this October, we will no longer be accepting industry support for our banquet, and I wanted to take this opportunity to explain how this change came about.

Due to their concerns about a recent focus on ethics in the VA, several of our colleagues went directly to General Counsel at their facility to ask for an opinion as to whether or not there were any ethics violations involved in their attending the annual NAVAO banquet. Specifically, their concern stemmed from the corporate sponsorship we have received for funding of the dinner for the past several years. Unfortunately, despite the fact that the dinner is not limited to VA Optometrists, nor do any of us directly (or indirectly) engage in negotiating medications for the VA formulary, General Counsel's opinion was that attending a dinner funded in part by a pharmaceutical company that does business with the VA would constitute an ethics violation. The opinion of General Counsel at that facility was then made known to me by one of the optometrists at that facility. Based on my awareness of this opinion, I feel it would be irresponsible and unwise for NAVAO to continue to accept industry funding.

Now there are really two ways to look at the situation. The first is to be extremely annoyed at these colleagues of ours who have apparently ruined a really great situation for NAVAO, and to want to string them up by their toes. This opinion has already been expressed to me by several of our colleagues.

The second way to look at it is to be relieved that this potential ethics violation was brought to the attention of the NAVAO leadership before it caused a more serious issue for the organization as a whole.

More

President's Report – cont.

I would argue that the latter is the more productive and useful way to view the situation, and that we accept it and move forward from this point. . In fact, until only recently, the NAVAO banquet had been financed entirely by its members, so I don't forsee any real difficulties with returning to something we had done for many years previously.

The NAVAO leadership will now focus on the situation that currently exists. We are confident that the NAVAO banquet will continue to be a wonderful event, and a great opportunity for camaraderie and fellowship

I would like to reiterate that this decision to decline further industry funding for our dinner was not a calculated or premeditated decision on the part of the NAVAO leadership, but is really more a reflection of the times we now live and have to work in. I would also like the membership to know that this change in now way reflects upon Alcon's support of NAVAO; in fact Alcon was quite disappointed to find that they could no longer sponsor the dinner.

As we develop plans for this years' dinner, we will certainly keep in mind budgetary considerations, and will do our best to make it an event that all will find affordable to attend.

And finally our deepest sympathies to the family of Dr. Raymond Adam, a VA colleague, on his untimely passing. A contribution from NAVAO has made to Adrian Raymond's college savings fund.

As requested by his widow, Libby Raymond, any individual donations can be sent directly to:

USAA IMCO
P.O. Box 659453
San Antonio, TX 78265

Checks should be made payable to: USAA College Savings Plan. It is very important that you include Adrian's account number on the check: 500611133-01.

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No Change in Drug Label Expected

The macular degeneration drug Lucentis (ranibizumab) may increase the risk for stroke at the FDA-approved dose, the manufacturer has announced. Patients receiving 0.5 mg of the drug had a 1.2% risk for stroke compared with a 0.3% risk for patients receiving 0.3 mg -- a significant difference -- after about 8 months of follow-up in an ongoing clinical trial. In a letter to physicians, the company reported that patients with a history of stroke faced a higher risk. However, researchers observed no significant increase in the rate of heart attack or vascular-related death between the two doses. No change in the drug's label

was expected, a company spokesman told the New York Times, because it already carries a warning about the risk for thromboembolic events.

http://firstwatch.jwatch.org/cgi/content/short/2007/129/2?rss=1

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Yet More Solution Being Recalled

Bausch & Lomb, which has been struggling to recover from a sweeping recall of its ReNu with MoistureLoc contact lens solution, said Tuesday it had initiated a recall of certain lots of its ReNu MultiPlus contact lens solution.
The solution was made at the company's plant in Greenville, South Carolina. Bausch (Charts) said the solution contained an elevated level of trace iron, which may result in discoloration of the solution in some bottles and shorten the shelf life of the product.

The company said it had received no reports of serious problems associated with the affected product. It believes virtually all of the affected product, manufactured about a year ago, has already been used by consumers. Bausch & Lomb said the recall involved 12 lots of the product. About 1 million bottles of solution from nine of the 12 lots were originally distributed in the United States.

Product from the 12 affected lots was also distributed in Canada, Latin America, Korea and Taiwan, where it is also being recalled. Bausch said it had notified the U.S. Food and Drug Administration of its recall. The company advises consumers to discard any lens care solution that appears to be discolored. The recalled lots all carry the expiration date "2008 - 03" on the bottle.
The company said it did not expect the costs associated with the recall to have a significant impact on its financial results.

http://money.cnn.com/2007/03/06/news/companies/bc.bausch2.reut/index.htm?cnn=yes

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On Its Way to Nevada Governor for Final Approval

SANTA FE (AP) - The Legislature has given final approval to a bill that would allow optometrists to perform certain surgical procedures. The measure now goes to Gov. Bill Richardson for his signature.

The legislation sparked an aggressive broadcast advertising campaign by ophthalmologists, who opposed the measure. Supporters contend that the proposed procedures, such as removing lesions from an eyelid, are relatively minor and optometrists have been doing them for years because they were not previously considered surgery. Ophthalmologists are physicians who specialize in eye diseases. They say superficial lesions can be serious, invasive cancer

and that optometrists lack the training to diagnose or treat the problem with techniques requiring the use of a scalpel.

http://www.kob.com/index.cfm?viewer=storyviewer&id=31088&cat=4HEALTH

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Decreased Myopia in Progressive-Wearing Kids

The present study investigated the relationship between parental refractive error and myopia progression in their offspring and the interaction between parental ametropia and the effects of wearing progressive-addition (PALs) or single-vision (SVLs) lenses on the progression of myopia in children enrolled in the Correction of Myopia Evaluation Trial (COMET).

The progression of myopia in a subset of COMET children (N= 232; 49% of initial group) was defined as the difference in mean spherical equivalent refraction of both eyes obtained by cycloplegic autorefraction between the baseline and 5-year visit. Parental refractions were obtained by noncycloplegic autorefraction (81%) or from recent eye examination records (19%).

The number of myopic parents (mean spherical equivalent refraction ≤–0.75 D) was directly related to myopia progression among children wearing SVLs: myopia in children with no (zero) myopic parents progressed (mean ± SE) –1.81 ± 0.18 D and with one myopic parent, –2.04 ± 0.13) D; these amounts were significantly less than the progression of children with two myopic parents (–2.59 ± 0.19 D). In the PAL group, progression was not significantly related to the number of myopic parents and was –2.01 D overall. Among children with two myopic parents, progression was –2.00 D in the PAL group, significantly less than the progression of children wearing SVLs (P = 0.03). Among children with zero or one myopic parent, progression did not differ significantly between the lens groups. When the data were adjusted for covariates, the interaction between treatment effect and number of myopic parents was significant (P = 0.01). Over the 5-year study period, axial length increased 0.93 ± 0.07 mm in children with two myopic parents wearing PALs and 1.18 ± 0.07 mm in children with two myopic parents wearing SVLs (P = 0.01). The axial length increase in children wearing SVLs and with two myopic parents was significantly more than the 0.89 ± 0.07 mm increase in children wearing SVLs and with zero myopic parents (P = 0.015).

Conclusions: Parental refraction was related to myopia progression and changes in axial length. Among COMET children with two myopic parents, myopia progression and increases in axial length were slower in the group wearing PALs than in those wearing SVLs, by a statistically significant but clinically minor amount. Because this study was ancillary to COMET and the present analyses are based on a subset of participants, conclusions must be regarded as suggestive.

http://www.iovs.org/cgi/content/abstract/48/2/562

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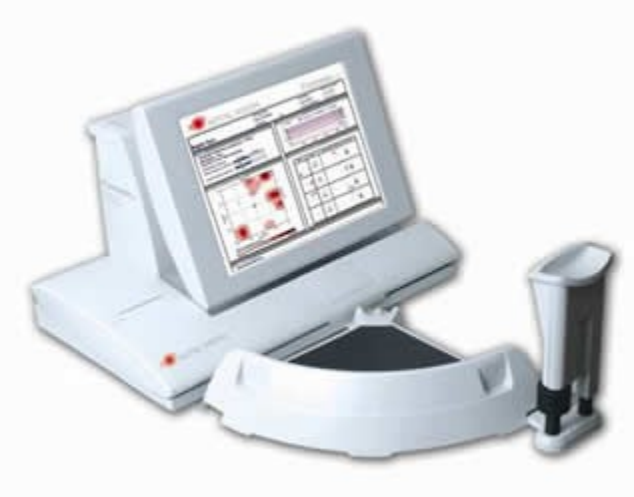
Device May Detect Conversion to Wet AMD

This clinically validated diagnostic device has FDA clearance for monitoring the progression of AMD and detecting the conversion from "dry" to "wet" AMD signaled by the onset of CNV. It's a second-generation device that offers a number of improvements to the first-generation Preview PHP, that the company says makes AMD monitoring and report interpretation quicker and easier.



The device generates a non-invasive eye exam that allows you to identify elevations in the patient's retinal pigment epithelium (RPE) and the bowing of the photoreceptor layer — both consistent with conversion from an intermediate to an advanced AMD stage, or "wet" AMD.

This eye exam is based on Vernier acuity, also known as hyperacuity, or the ability to perceive small differences in the spatial relationships of two objects. Vernier acuity is ten times more sensitive than standard visual acuity.2 Through its use of hyperacuity, the Foresee PHP can overcome the brain's ability to compensate for small visual field defects and may identify CNV lesions prior to the patient experiencing any significant vision loss.

http://www.optometric.com/article.aspx?article=71852

http://www.foreseephp.com/Eng/Products/Professionals/Foresee.asp

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Games Sharpen Vision 20 Percent

Video games that contain high levels of action, such as Unreal Tournament, can actually improve your vision. Researchers at the University of Rochester have shown that people who played action video games for a few hours a day over the course of a month improved by about 20 percent in their ability to identify letters presented in clutter - a visual acuity test similar to ones used in regular ophthalmology clinics.

"Action video game play changes the way our brains process visual information," says Daphne Bavelier, professor of brain and cognitive sciences at the University of Rochester. "After just 30 hours, players showed a substantial increase in the spatial resolution of their vision, meaning they could see figures like those on an eye chart more clearly, even when other symbols crowded in."

Bavelier and graduate student Shawn Green tested college students who had played few, if any, video games in the last year. "That alone was pretty tough," says Green. "Nearly everybody on a campus plays video games."

At the outset, the students were given a crowding test, which measured how well they could discern the orientation of a "T" within a crowd of other distracting symbols - a sort of electronic eye chart. Students were then divided into two groups. The experimental group played Unreal Tournament, a first-person shoot-'em-up action game, for roughly an hour a day. The control group played Tetris, a game equally demanding in terms of motor control, but visually less complex.

After about a month of near-daily gaming, the Tetris players showed no improvement on the test, but the Unreal Tournament players could tell which way the "T" was pointing much more easily than they had just a month earlier.

"When people play action games, they're changing the brain's pathway responsible for visual processing," says Bavelier. "These games push the human visual system to the limits and the brain adapts to it. That learning carries over into other activities and possibly everyday life."

The improvement was seen both in the part of the visual field where video game players typically play, but also beyond - the part of your vision beyond the monitor. The students' vision improved in the center and at the periphery where they had not been "trained." That suggests that people with visual deficits, such as amblyopic patients, may also be able to gain an increase in their visual acuity with special rehabilitation software that reproduces an action game's need to identify objects very quickly.

The team is now delving into how the brain responds to other visual stimuli. They plan to use what would be a video gamer's dream: a new 360-degree virtual-reality computer lab now being completed at the University of Rochester.

This research appears next week in the journal Psychological Science, and was funded by grants from the National Institutes of Health.

http://www.medicalnewstoday.com/medicalnews.php?newsid=62412

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Children's Vision Being Destroyed, Site Says

http://www.myopia.org/index.htm

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Patanol Now Available as Once-a-day Dose

Patients are beginning to present with their seasonal allergic symptoms. Several changes have come about since last year. Patanol is now available as 0.2% once-daily dosing; and Zaditor is now available OTC. Below is a link to a "review-type" article that may be informative.

http://www.eyeupdate.com/pages/occular_allergy/ocular_allergy_medicine_profile.html

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Thirty-six Percent of SITA Defects Missed By Matrix

The purpose of this study was to compare visual field (VF) defects found by Swedish interactive thresholding Algorithm (SITA) perimetry and Matrix perimetry, a new VF device that utilizes frequency doubling technology in a 24-2 test pattern.

Fifty eyes from 50 subjects with SITA field defects were recruited for an observational study. Swedish Interactive Threshold Algorithm and Matrix VF testing were performed on patients from a glaucoma practice. To evaluate the learning effect on the performance of the VF, we tested subsets of each group who had previous experience with standard automated perimetry (SAP).

Test duration was significantly shorter for Matrix (SITA, 357.0±85.6 seconds; Matrix, 319.5±16.5 seconds; P = 0.0002, paired t-test). Thirty-six percent of eyes with SITA VF defects showed a normal Matrix field. In 30 of 32 eyes (94%) where both devices showed VF defects, the defects were congruent. Mean threshold value was significantly lower with Matrix compared to SITA (P<0.0001, paired t-test), as was MD (−5.34±5.42 dB, −4.14±5.29 dB, respectively; P = 0.03, paired t-test). There was no significant difference in PSD between the 2 devices (P = 0.78, paired t-test). Matrix delineated significantly smaller (P = 0.005, Wilcoxon's test) and deeper (P<0.001, Wilcoxon's test) defects than those found with SITA. Similar results were observed in the subgroups with prior SAP experience.

Conclusions

The Matrix examination did not detect 36% of abnormal SITA fields. Matrix field defects were smaller and deeper than those appearing in SITA perimetry.

http://www.ophsource.org/periodicals/ophtha/article/PIIS0161642006010608/abstract

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Cigarette Smoking, *CFH*, *APOE*, *ELOVL4*, and Risk of Neovascular Age-Related Macular Degeneration

Researchers attempted to examine if the genes encoding complement factor H (CFH), apolipoprotein E (APOE), and elongation of very-long-chain fatty acids–like 4 (ELOVL4) confer risk of neovascular age-related macular degeneration (AMD) in an independent or interactive manner when controlling for smoking exposure.

They studied 103 unrelated patients with neovascular AMD who each had at least 1 sibling with normal maculae. Smoking histories were obtained. Genotyping was performed by analyzing amplified genomic fragments from CFH, APOE, and ELOVL4 by direct sequencing or by restriction endonuclease digests. Conditional logistic regression analysis was used to build a multifactor model.

Results: For CFH, only the CC genotype carried a statistically significant elevation of disease risk (odds ratio, 49.37; 95% confidence interval, 6.20-393.22; P<.001). No significant association was observed between neovascular AMD and APOE or ELOVL4. No significant interactions were found between smoking and having the CFH or APOE genotype nor were significant interactions found between the CFH, ELOVL4, and APOE genotypes.
Conclusions: Smoking and having the CFH CC genotype independently increase risk of neovascular AMD. APOE and ELOVL4 genotypes do not seem to modify risk. Smoking 10 pack-years or more and having the CFH CC genotype increase one's risk of neovascular AMD 144-fold compared with smoking less than 10 pack-years and having the CT or TT genotype.

http://archopht.ama-assn.org/cgi/content/abstract/125/1/49

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Level II Codes and Guidelines Released

From the AOA's EBCB:

A New Era in Physician Performance Quality Measurements
New Level II CPT Codes
by Rebecca H. Wartman OD

Beginning in January 2007, new Level II CPT codes have been developed as supplemental tracking codes to be appended to claims for certain medical conditions. These new Level II CPT codes will be used in the future to determine the quality of the care an individual provider gives to patients with certain disease processes. Ultimately, reimbursement amounts may be tied to the quality of care a provider gives to patients.

More information on all of these new measures will be published as it becomes available. Beginning in July 2007, Medicare will ask for voluntary use of some or all of these Level II CPT codes to track performance. According to the CMS Physician Quality Reporting Initiative (PQRI) guidance, if at least three of these measures are used at least 80% of the time, a provider will receive a lump sum bonus payment of 1.5% on all of their Medicare claims for the reporting period of July 1, 2007-December 31, 2007.

Suffice it to say that a new era is dawning in physician performance quality measures and all optometrists will be expected to comply along with all of the other health care providers. Happy coding....
While no reimbursement is included, these Level II codes tell the insurers when certain aspects of care are performed and should aid in reducing the need for actual claims review. Currently the Level II CPT codes cover aspects of eye care for diabetes, glaucoma, age related macular degeneration, and cataracts.

The process is simple. The insurance claim form is completed in the normal fashion with the professional services coded on the first line of the CMS claim form with the proper disease ICD-9 diagnosis code. The next line(s) will be

the Level II CPT code or codes applicable for the disease ICD-9 diagnosis code and a zero dollar amount.

Although the use of these codes is voluntary, the AOA hopes all optometrists who file insurance claims will begin to use these new codes.

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Help Keep Us Informed

Please don't hesitate to submit news and notes to the Optimum VA. The more you submit, the better our newsletter will be. Such information may include:

- Letters to the editor
- Case reports
- Photos
- Article abstracts (include publication information)
- Upcoming events (CE, meetings, etc.)
- Personal accomplishments
- Internet links

*** Feel free to submit at any time by clicking the link Contact Optimum VA which is also located on the front page in the Editor's Box. Submission and publication dates are listed below.**

**** Residents and students are also encouraged to submit.**

Issue	Submissions Due	Publication Date
Winter	December 15	January 1
Spring	March 15	April 1
Summer	June 15	July 1
Fall	September 15	October 1

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Associations, Institutes, Organizations, Societies

All About Vision
Alliance for Aging Research
American Academy of Ophthalmology
American Academy of Optometry

American Academy of Pediatrics
American Optometric Association
American Optometric Foundation
Association of Regulatory Boards of Optometry (ARBO)
Council for Refractive Surgery Quality Assurance
Eye Advisory
Eye Surgery Education Council
Glaucoma Research Foundation
Healthy Vision 2010
International Glaucoma Association
NASA Vision Group
National Eye Research Foundation
National Keratoconus Foundation
National Optometric Association
NBEO
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World Council of Optometry

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Coding

AHIMA
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Centers for Medicare & Medicaid Services
Healthcare Common Procedure Coding System (HCPCS)
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- Alabama
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Bausch & Lomb
British Contact Lens Association
CIBA Vision NIGHT & DAY
CIBA Wesley-Jessen
Contact Lens and Anterior Eye
Contact Lens Council
Contact Lens Manufacturers Association
Contact Lens Spectrum
CooperVision
Innovative Sclerals Ltd.
International Association of Contact Lens Educators (IACLE)
New Zealand Contact Lens Society
Ocular Sciences
Official Site of Silicone Hydrogel Lenses
Virtual Consultant

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Heart of America Contact Lens Society
Midwest Vision Congress & Expo
OptoEast
OptoWest
Southern Council of Optometrists
Vision Expo East
Vision Expo West

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Foreign Web Sites

American Academy of Optometry (British Chapter)
Association of Optometrists, UK
Australasian College of Behavioural Optometrists (ACBO)
Bradford, University of, Department of Optometry (UK)
Brazilian Optometry Association
Canadian Association of Optometrists
College of Optometrists - UK
Eye Health Council of Canada
Hong Kong Polytechnic University Optometry Section (PRC)
Hong Kong Society of Professional Optometrists
Institute of Optometry - UK
Karolinska Institue - Sweden
Melbourne College of Optometry - Australia
New Zealand Association of Optometrists

Ontario Association of Optometrists
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Pacific University
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UAB School of Optometry
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AskPhysicians.com
National Headache Foundation
Sjögrens Syndrome Foundation

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REVIEW OF THE PHYSIOLOGY OF NARCOTIC ANALGESICS

by Steven Mordukowitz,OD,FAAO, Bronx VAMC

With oral pharmaceutical certification now prevalent in most states, a good understanding of the physiology of the opioid receptors and pain pathways is imperative for prescribing narcotic analgesics.

The "Gate Control Theory of Pain" has been one of the most prominent explanations of pain control mechanisms. This theory describes how incoming afferent pain impulses are controlled by a series of inhibitory inter-neurons in the spinal chord (i.e.- substantia gelatinosa) and from descending anti-nociceptive pathways from the higher brain centers (such as the reticular formation and peri-aqueductal/periventricular gray matter). This gating mechanism can prevent painful stimuli from being passed on from the spinal chord to the higher brain centers. Many of the neurons in this pathway have opiate receptors, which when stimulated can enhance the gate and inhibit painful stimuli from reaching the CNS.

Details of the gate theory are as follows (see Fig #1):
A-fibers, also known as "Pressure fibers", are thick myelinated fast-conducting fibers that enhance the firing of the substantia gelatinosa, therefore reducing pain. C-fibers, also known as "Pain fibers", are thinner unmyelinated slow-conducting fibers that inhibit the actions of the substantia gelatinosa, thereby increasing pain sensation. Once activated, the C-fibers themselves stimulate the dorsal horn cells of the spinal chord, which then transmit ascending pain impulses to the brain.

Opiate receptors are present all over the body, particularly in the pain pathways and the gastro-intestinal tract. Examples of endogenous opiates include the enkephalins and endorphins.

Important pain mediators released by damaged tissue following injury include intracellular Potassium (where pain intensity can be correlated to the amount of extracellular local K+ concentration), Histamine, Seratonin and Prostaglandins. Other prominent pain mediators include Bradykinin (released from circulation) and Substance-P (released from nerve endings).

Affects of Narcotic Analgesics on the Pain Pathway:

The sensation of pain can be modified by a number of different mechanisms at various locations in the pain pathway: (see Fig 2)

1. By reducing the sensitivity of the peripheral nociceptors by blocking the actions of prostaglandins released by the injured tissues.

NSAIDs are commonly used for this purpose. Additionally, Local Anesthetics are also effective in blocking the pain pathway at the nociceptor level as well as at their sensory nerve axons.

2. By decreasing the transmission of nociceptive impulses in the dorsal horn cells of the spinal chord.

Narcotic Analgesics are effective in relieving pain by selectively binding to the opioid receptors in the spinal column (PNS) and the Thalamus (CNS).

3. By inhibition of pain perception in the cerebral cortex (CNS).

Narcotic Analgesics and General Anesthetics are effective in producing analgesia by their actions in the CNS.

4. By altering the emotional responses to pain.

The Anti-depressants act as "co-analgesics" along with other central and peripheral nervous system acting agents (ie- opioids, etc.) to alleviate discomfort.

Actions of Narcotic Analgesics:

Narcotic Analgesics (or "Opioids") are described as drugs with "morphine-like" actions, which relieve pain by selectively binding to opioid receptors in the CNS and spinal chord. In addition to producing analgesia, the opioid analgesics have been known to cause a number of side and adverse effects, including constipation and respiratory depression, which can be life threatening.

Narcotic Analgesics function by producing a number of depressive as well as stimulatory effects:

DEPRESSIVE EFFECTS:

1. Analgesia:

Effects of analgesia include reducing the sensation of pain without loss of consciousness. Some common side effects include confusion, dizziness, drowsiness, reduced concentration and attention, mood alterations (ie- euphoria or depression) and seizures with high doses.

2. Anti-Tussive actions:

Antitussive effects of narcotic analgesics involve depression of the cough reflex by inhibiting the cough-control center in the brainstem.

3. Respiratory Depression:

This is the most dangerous adverse effect of narcotic agents. Severe respiratory depression results from reduced responsiveness of medullary chemoreceptors to increase CO_2 levels in the blood.

STIMULANT EFFECTS:

1. Analgesia:
Opioids stimulate the anti-nociceptive pathways (ie- reticular formation, paraventricular gray matter, etc.) to relieve pain.

2. Nausea and Vomiting:
Narcotic agents can directly stimulate the *Chemoreceptor Trigger Zone* in the brainstem causing nausea and vomiting.

3. Cardiovascular Effects:
Narcotic agents can dilate the resistance capacitance vessels of the body resulting in orthostatic hypotension. The increase CO2 retention produced by these agents also leads to cerebral vasodilation, leading to increased intra-cranial pressure.

4. Pupil Miosis:
This response is due to stimulation of the Edinger Westphall (CN III) nucleus in the brainstem.

5. Constipation/Anti-Diarrheal effects:
Opioids increase smooth muscle tone in the GI tract, thereby reducing peristalsis and slowing digestion, leading to spastic constipation. Constipation is the most common side effect encountered with the Opioid Analgesics.

6. Decrease Urine Output:
Additionally, these agents reduce urine output by increasing bladder sphincter tone, as well as increase Anti-Diuretic Hormone (ADH) release, thereby increasing water reabsorption.

CONTRAINDICATIONS:

Contraindications to narcotic analgesics include:

1. CNS:
CNS effects include a history of head trauma, convulsions, alcohol abuse and use of CNS acting agents (ie- amphetamines, MAO inhibitors, phenothiazines).

2. Asthma/COPD:
This is the leading restriction for use of narcotic analgesics.

3. Liver or Renal disease:
Narcotic Analgesics should be avoided in liver or renal disease due to their effects on metabolism and excretion.

4. Allergy to Opioids.

NARCOTIC ANALGESIC AGENTS:

A flow sheet illustrating the clinical potency of Narcotic Analgesics is included in Fig #3.
This can be used as a guideline in prescribing the various opioid agents in a clinical setting. A brief review of each of the individual narcotic agents is also included in the ensuing paragraphs.

Morphine: (Schedule 2)
Morphine is the prototype analgesic that binds to opiate receptors in the CNS and spinal chord. It is effective for severe post-operative pain or for chronic pain associated with malignancy. Morphine produces analgesia without loss of consciousness. Tolerance and addiction can result from chronic use.

MORPHINE RECEPTOR AGONISTS:

1. Codeine: (Schedule 3)
Codeine is a weaker opiate, approximately 1/6th Morphine's potency, with similar efficacy. It can therefore be used longer than other narcotics since it has fewer side effects and a lower addiction potential. In addition to analgesia, Codeine possesses anti-tussive actions. Codeine is commonly prescribed in combination with aspirin (Empirin) or acetaminophen (Tylenol 3).

2. Hydromorphone: (Schedule 2)
Hydromorphone is a much stronger analgesic agent, approximately 10x as potent as Morphine. It has a high abuse potential, since it causes significant euphoria. It is therefore classified as a Schedule 2 Opioid.

3. Oxycodone: (Schedule 2)
Oxycocone is a very potent analgesic, approximately 12x more potent than Codeine, used for moderate to severe pain. It is available in mixtures only (ie- Percodan= with aspirin, Percocet= with acetaminophen). This is an orally effective drug, with almost complete absorption, with equal potency to injected morphine. Oxycodone produces significant euphoria, therefore its potential for abuse and addiction is quite high.

4. Hydrocodone: (Schedule 3)
Hydrocodone is commonly used for out-patient pain management; it is 6x more potent than codeine. It is often the preferred agent in eye care for short-

term pain management. It is commonly prescribed in combinations with acetaminophen (Vicodin) and Ibuprofen (Vicoprofen). Hydrocodone produces less sedation and constipation than codeine, but more euphoria.

5. Meperidine (Demerol): (Schedule 2)
Meperidine produces analgesia only. It has no anti-tussive or anti-diarrheal actions. Meperidine has 1/5 of Morphine's potency with equal efficacy. It has been used for treatment of post-operative pain and to produce sedation or support of anesthesia in adults and children. Meperidine is more effective parenterally and produces brief analgesia with significant euphoria.

5. Methadone: (Schedule 2)
Methadone has been used in Narcotic Maintenance programs in treating patients with opioid addiction. It is an excellent analgesic with equal potency but longer duration of action than Morphine.

6. Propoxyphene: (Schedule 4)
Propoxyphene is a weak acting Methadone analogue, commonly used for treatment of minor pain in the elderly. It produces significant sedation and is commonly prescribed in combination with aspirin (Darvon) and Acetominophen (Darvocet).

AGONIST/ANTAGONISTS:

1. Pentazocine (Talwin): (Schedule 4)
Pentazocine is the only oral acting partial agonist/weak antagonist narcotic. It is a weaker analgesic, approximately ¼ to ½ Morphine's potency, producing less respiratory depression and abuse potential than other opiates. Because of its low addiction, it has been used for chronic pain management.

PURE ANTAGONISTS:

1. Naloxone (Narcan):
Naloxone is a pure opioid antagonist (parenteral only) for short term treatment of respiratory depression induced by narcotic analgesics. It rapidly reverses the effects of narcotic analgesics. Being a pure antagonist, it has no significant actions of its own.

2. Nalorphone:
Nalorphone has both agonist and antagonist actions. When used alone, Nalorphone displays some weak opiate-like effects. It functions as a complete antagonist when used to counter the effects of narcotic analgesics.

CENTRALLY ACTING AGENTS:

1. Tramadol (Ultram):

Tramadol is a weaker oral analgesic agent used for treatment of mild pain. It produces analgesia without irritating the GI tract. It is not a controlled substance, since it produces no dependence or addiction.

SUMMARY:

Narcotic Analgesics are commonly used as an adjunctive treatment in the short-term management of ocular pain. They often display additive or synergistic effects when combined with Non-Steroidal Anti-Inflammatory Drugs (NSAIDs). They commonly can be used as safer alternatives for patients with contraindications to NSAIDs.

Peak affects of most of the narcotic analgesics are within one and a half to two hours. Therefore, it is imperative to advise patients to expect relief within this time period to avoid over dosage or misuse of these agents. Additionally, narcotic agents should not be given within 14 days of treatment with MAO inhibitors and other CNS acting agents.

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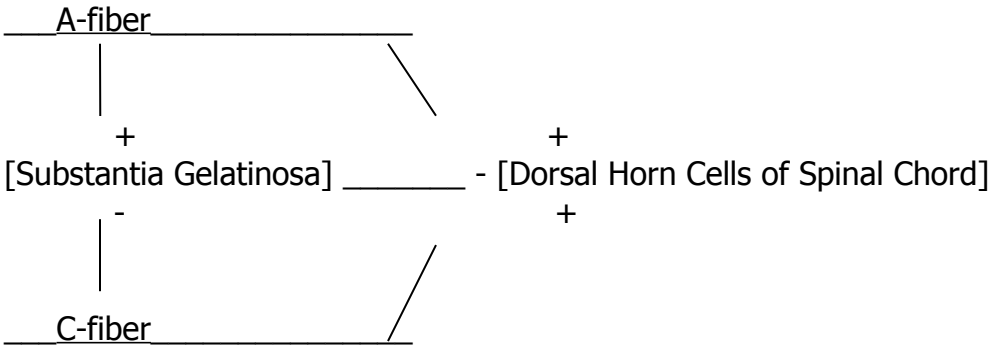
FIG #1: "GATE-CONTROL" THEORY OF PAIN:



FIG# 2: ACTIONS OF NARCOTIC ANALGESICS ON PAIN PATHWAYS:

Cerebral Cortex: *General anesthetics*

Thalamus: *Opioids*

Reticular Formation/posterior cortical areas: *Anti-depressants*

(descending anti-nociceptive pathway)

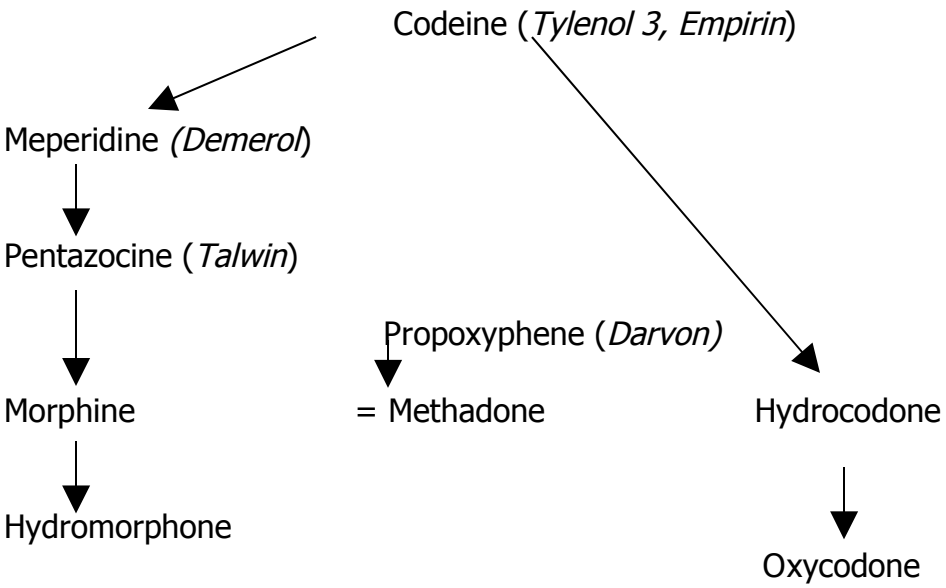
(spinothalamic tract)

Dorsal Horn cells of spinal chord: *Opioids*

Nociceptors: *Local Anesthetics*

Prostaglandins: *NSAIDs*

FIG #3: FLOWSHEET FOR POTENCY OF NARCOTIC ANALGESICS:



CNS:
Tramadol

Antagonists:
Naloxone (*Narcon*)
Nalorphone

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