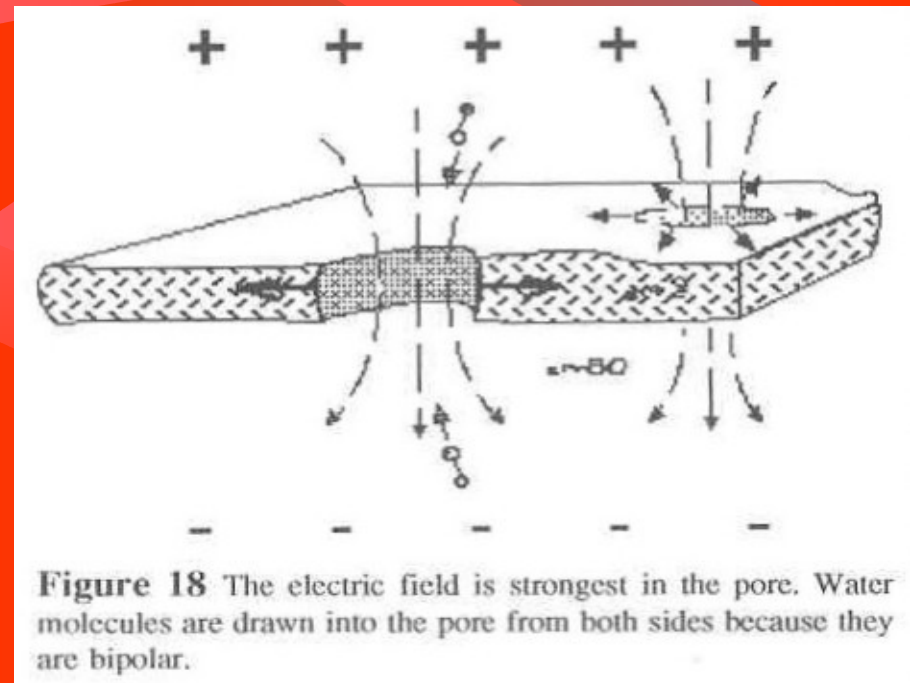
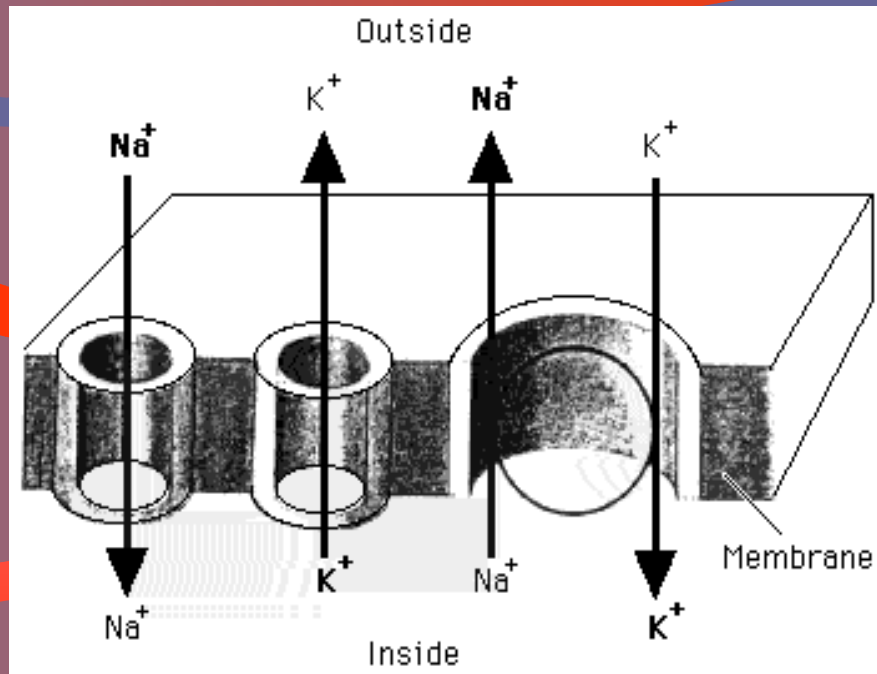
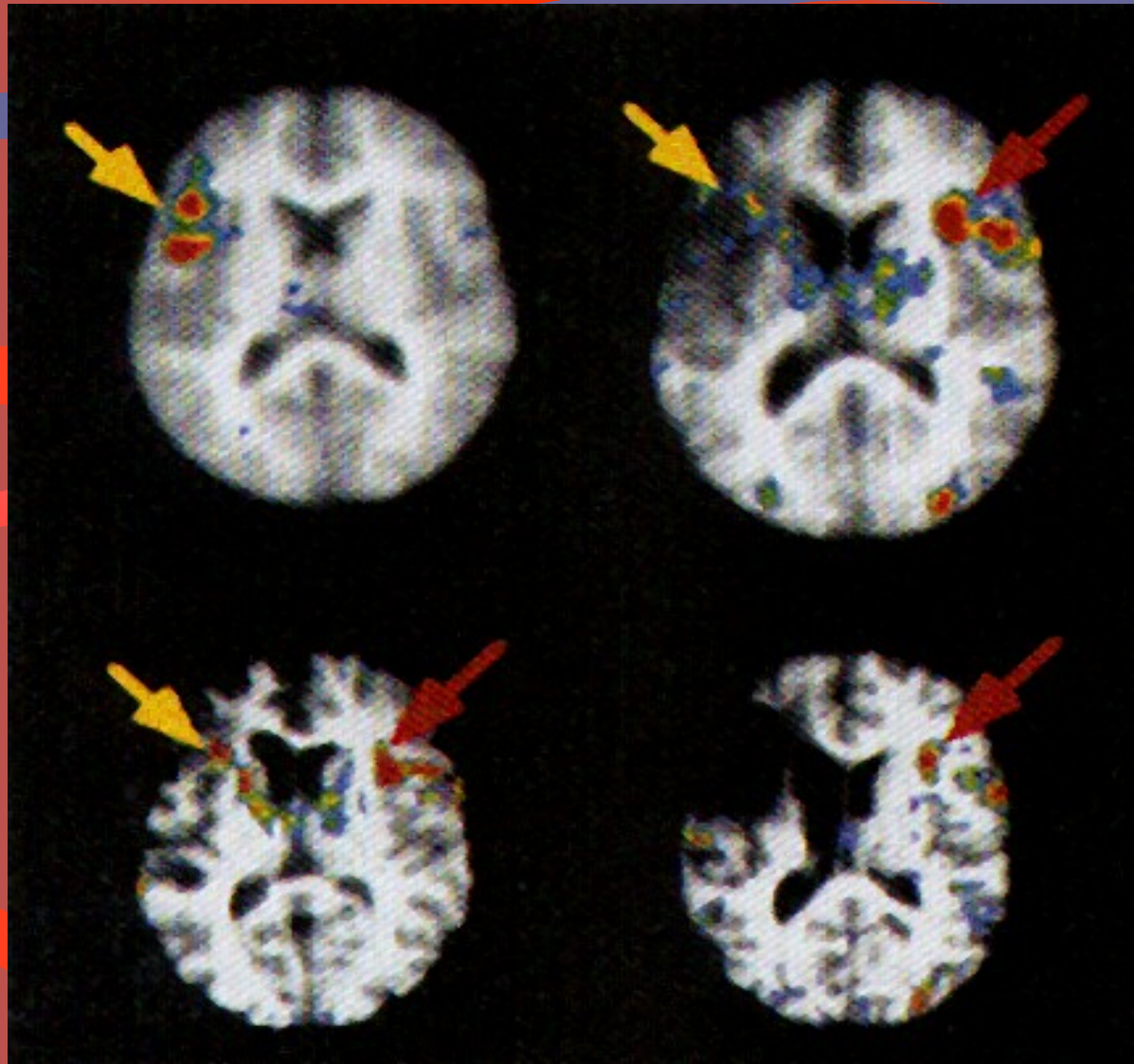


# Transmembrane Ionic Flow



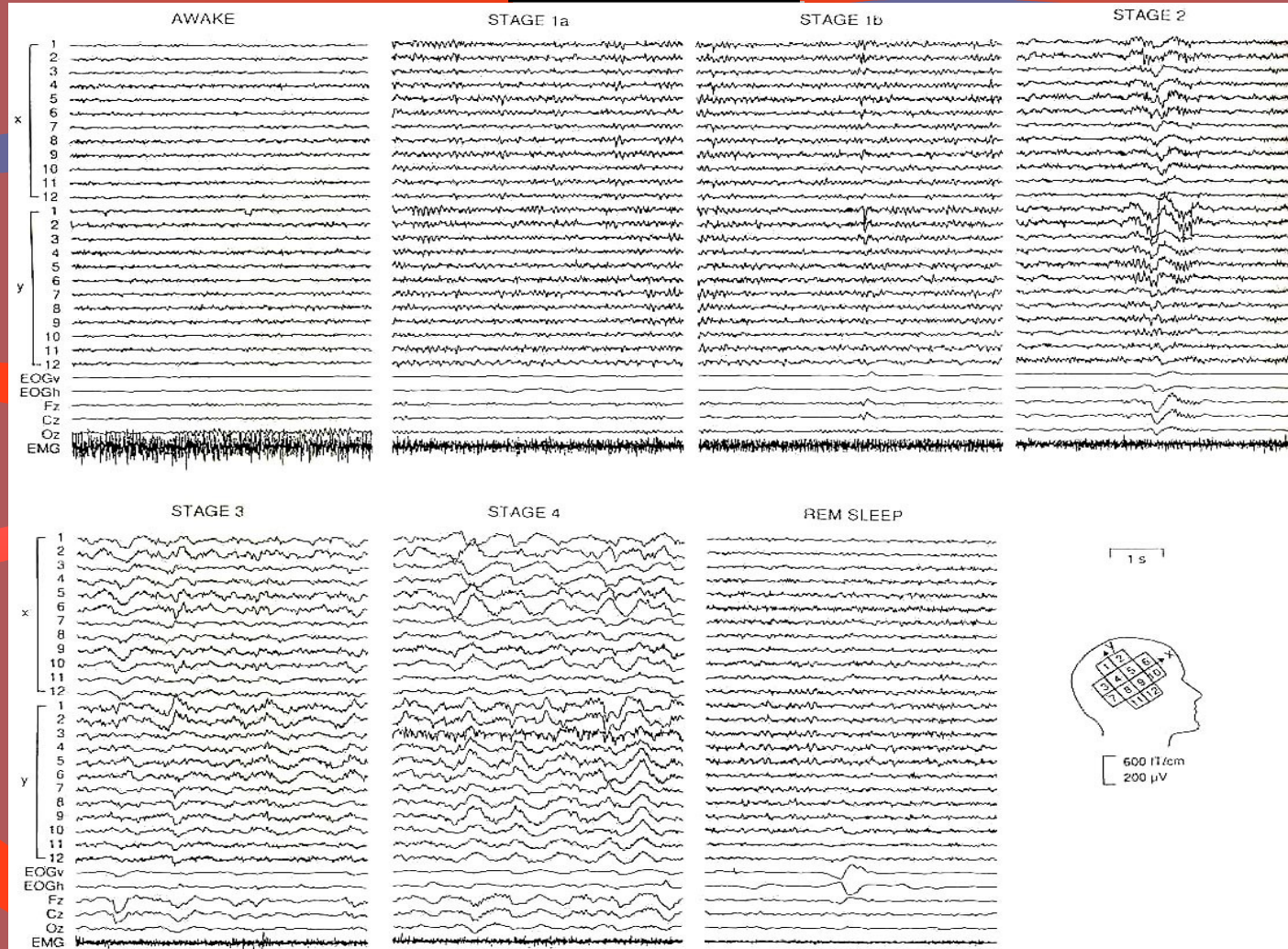
**Where there is flow, there is current → EMF!**



# The Brain

- 150 billion neurons.
- 100,000 synapses each.
- Still (many orders of magnitude) more complex than the most sophisticated computer.

# Electrical Activity in the Brain



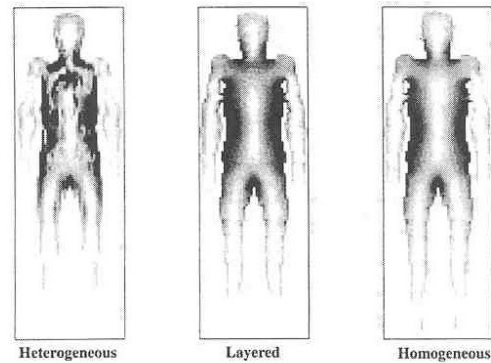
**Figure 58.18.** Different sleep stages in one subject. A typical V-wave occurs during Stage 1b. During Stage 2, light sleep, K-complexes occur both in MEG and EEG. In deep sleep, Stages 3 and 4, slow activity is seen in the whole measurement area. Note the eye movements and

the decreased EMG activity during the REM stage. (Adapted from Lu, S.-T., Kajola, M., Joutsiniemi, S.-L., Knuutila, J., and Hari, R. 1992. Generator sites of spontaneous MEG activity during sleep. *Electroencephalogr. Clin. Neurophysiol.* 82:182-196.)

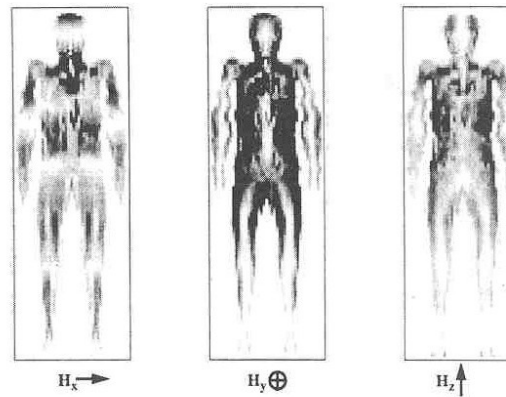


# Induced Currents

the computed quantities. The corresponding ratios of maximum computed current densities for the side-to-side exposure are 8.6, 27, and 3.1 respectively, indicating that simple weight-based scaling is likely inadequate.



**Figure 38:** Spatially-distributed induced current density profile in a mid-frontal plane of three models of man exposed to a 1 tesla field polarized back to front. Reproduced from Xi *et. al.* 1994.



**Figure 39:** Spatially distributed induced current density distribution in a mid-frontal plane of a heterogeneous man model exposed to a 60 Hz magnetic field oriented in the three orthogonal directions shown. Reproduced from Xi *et al.* 1994.

# Simple Electronic Diagram

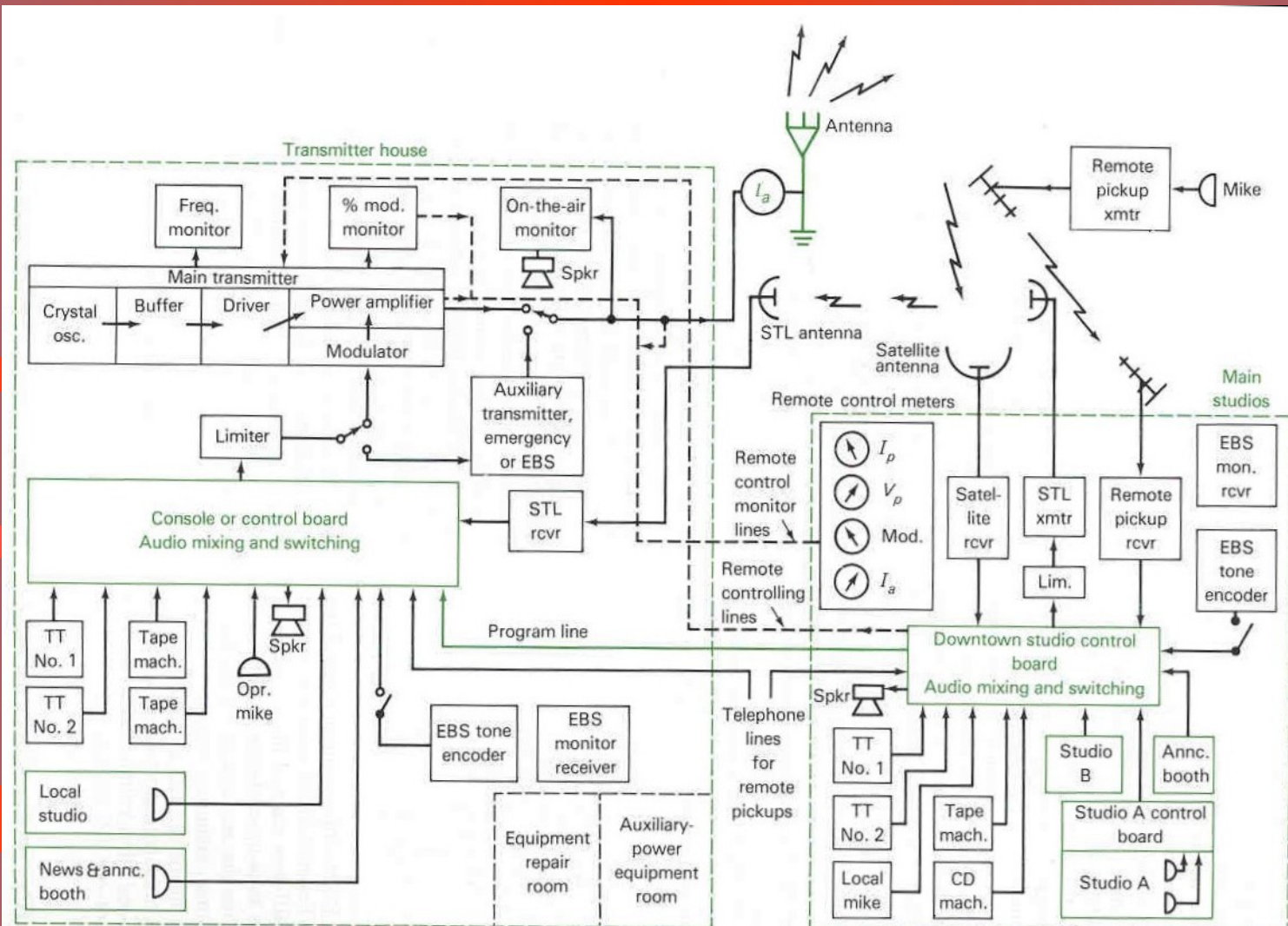


FIG. 24-1 Block diagram of an AM broadcast transmitting station, including STL and remote-pickup transmitter. Remote-control lines shown dashed.

# Biological Example from Nordenstrom

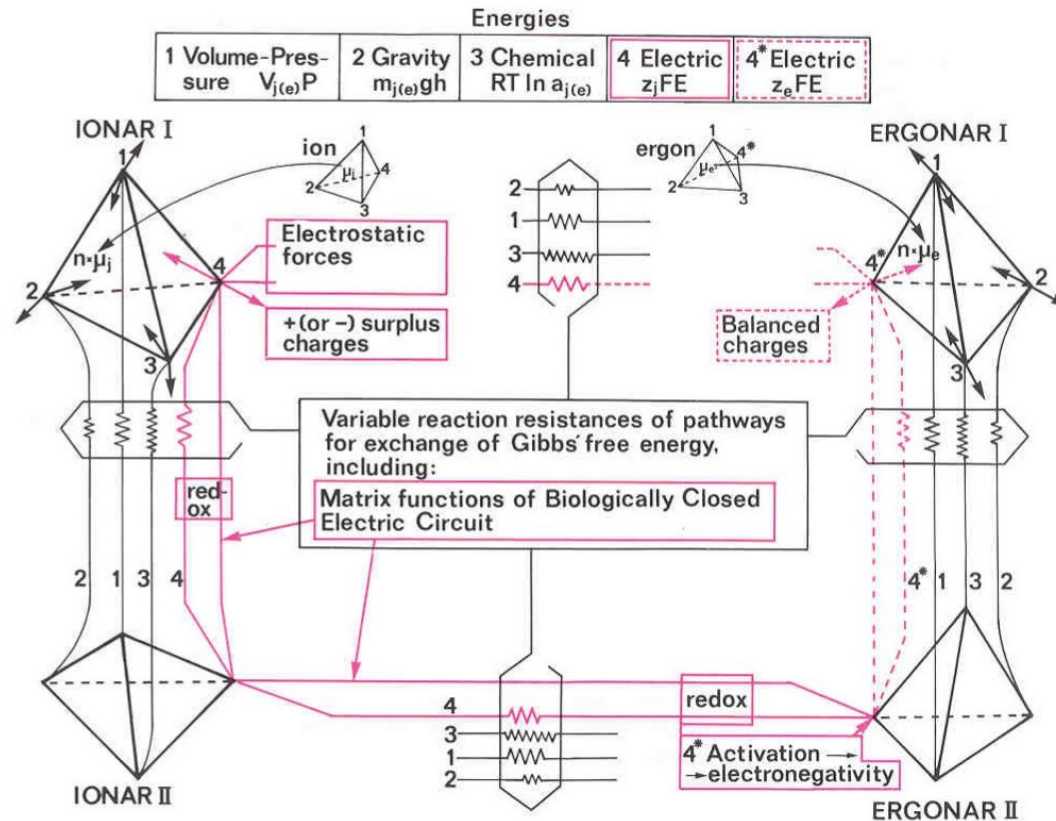
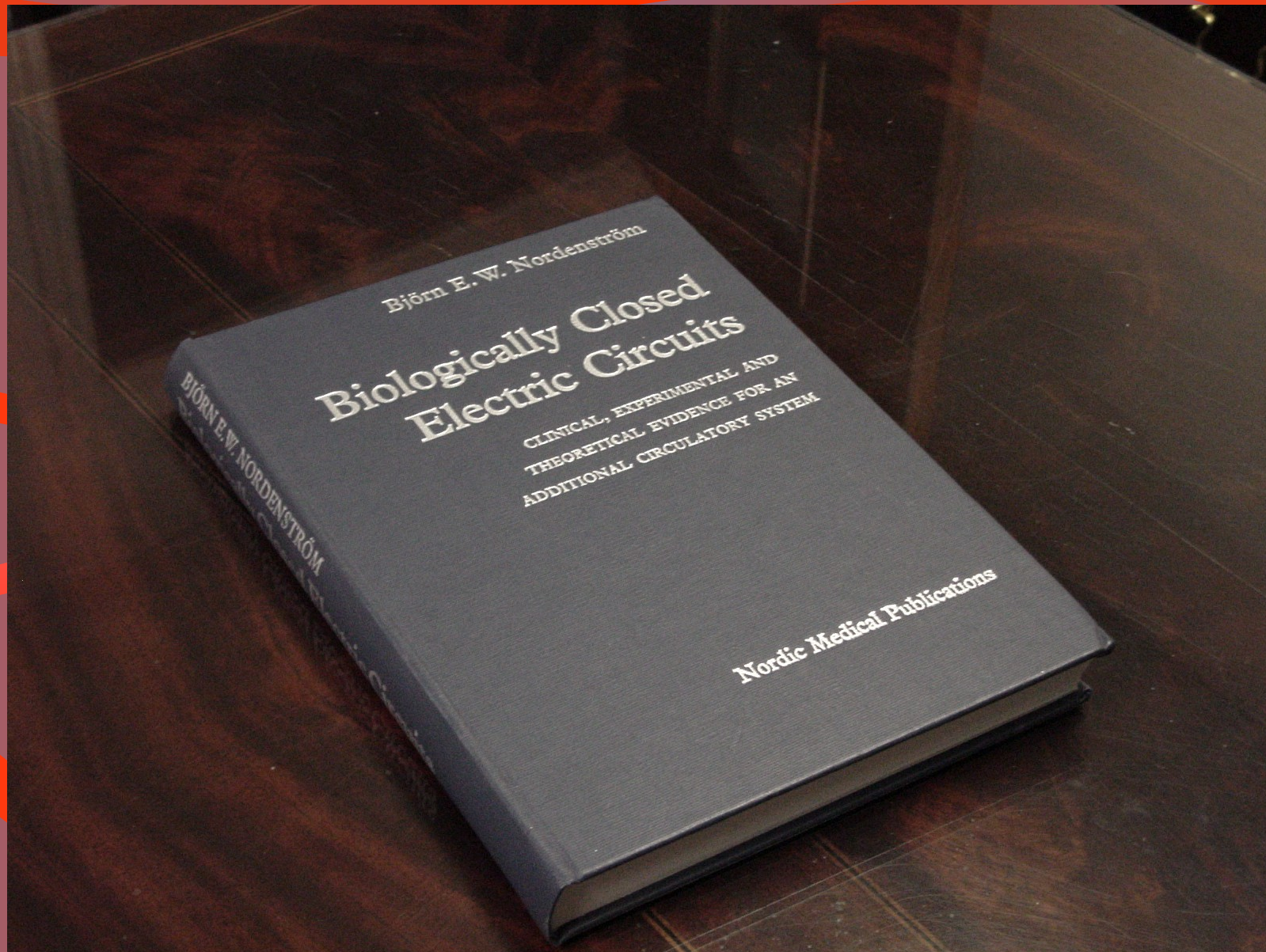


Fig. XIII:2. Principle of exchange of energy over BCEC systems. The quadripartite energy of ergonars and ionars is depicted graphically as tetrahedrons. The electrical system of BCEC circuits is depicted in red. Ions and ergons carry four energy factors (1-4). Ions carry a + or - surplus of immediately available electric energy (4) while an ergon carries balancing + and - charges (4\*). These ergonic charges may be brought into imbalance, leading to a varying degree of electronegativity. Collections of  $n$  ions or  $n$  ergons are repre-

sented by ionars and ergonars I and II. Factor 4\* permits ergonars (e.g., oxygen or glucose) to "save" their energy from reactions during transport until suitable conditions for energy exchange are available. Activation of factor 4\* is necessary for redox reactions of ergonars. The electromotive force of BCEC systems is directly dependent on factor 4 of ionars and indirectly on factor 4\* of ergonars.

For further explanation, see text.





Björn E. W. Nordenström

# Biologically Closed Electric Circuits

CLINICAL, EXPERIMENTAL AND  
THEORETICAL EVIDENCE FOR AN  
ADDITIONAL CIRCULATORY SYSTEM

Nordic Medical Publications

BJÖRN E. W. NORDENSTRÖM



# Principles

- It's a question of complexity.
- (Chaos of energy transfers).
  - **Origin - magnetic or electrical**
  - **Current flow**
  - **Timing**
  - **Storage = capacitance**
  - **Frequency and intensity**
  - **Delivery**

# **Principles**

- **Billions of minute EM generators**
- **Infinitely complex resultant patterns**
  - **Number**
  - **Alignment**
  - **Combinations**
  - **Type**
    - **Ionic flow**
    - **Electronic flow**
    - **DC or AC**
    - **Chemical bonds**
    - **Whatever changes electrical flow,**
    - **changes the Electromagnetic Fields**

# **Transcranial** **Stimulation**

- Increase neurotransmitters
- Increase catalysm
- Increase cyclic AMP use
- Reprogramming for normalization
- Peripheral stimulation influences



## **DL-phenylalanine markedly potentiates opiate analgesia - an example of nutrient/pharmaceutical up-regulation of the endogenous analgesia system**

**Russell AL, McCarty MF**, Brampton Pain Clinic, Bramalea, Ontario, Canada.

**In the author's clinical experience, concurrent treatment with DL-phenylalanine (DLPA) often appears to potentiate pain relief and also ease depression in patients receiving opiates for chronic non-malignant pain. An analysis of this phenomenon suggests that it may be mediated, at least in part, by up-regulation of the 'endogenous analgesia system' (EAS), a neural pathway that projects caudally from medullary nuclei to the dorsal horn of the spinal column; when stimulated by chronic pain or therapeutic measures such as opiates or acupuncture, the EAS suppresses activation of second-order pain-receptive neurons in the dorsal horn, and thereby alleviates pain. Since serotonin and enkephalins are key neurotransmitters in the EAS, it is reasonable to predict that measures which promote serotonin activity (such as 5-hydroxytryptophan and serotonin-reuptake inhibitors) as well as enkephalin activity (such as D-phenylalanine, an enkephalinase inhibitor) should potentiate EAS-mediated analgesia - a view consistent with much previous medical research. Comprehensive support of the EAS with well-tolerated nutrients and pharmaceuticals may amplify the analgesic efficacy of chronic opiate therapy, while enabling dosage reductions that minimize opiate side-effects. Analogously, this approach may complement the efficacy of acupuncture and other analgesic measures that activate the EAS. Copyright 2000 Harcourt Publishers Ltd.**

# Measured Consequences of Applied ? EMFs

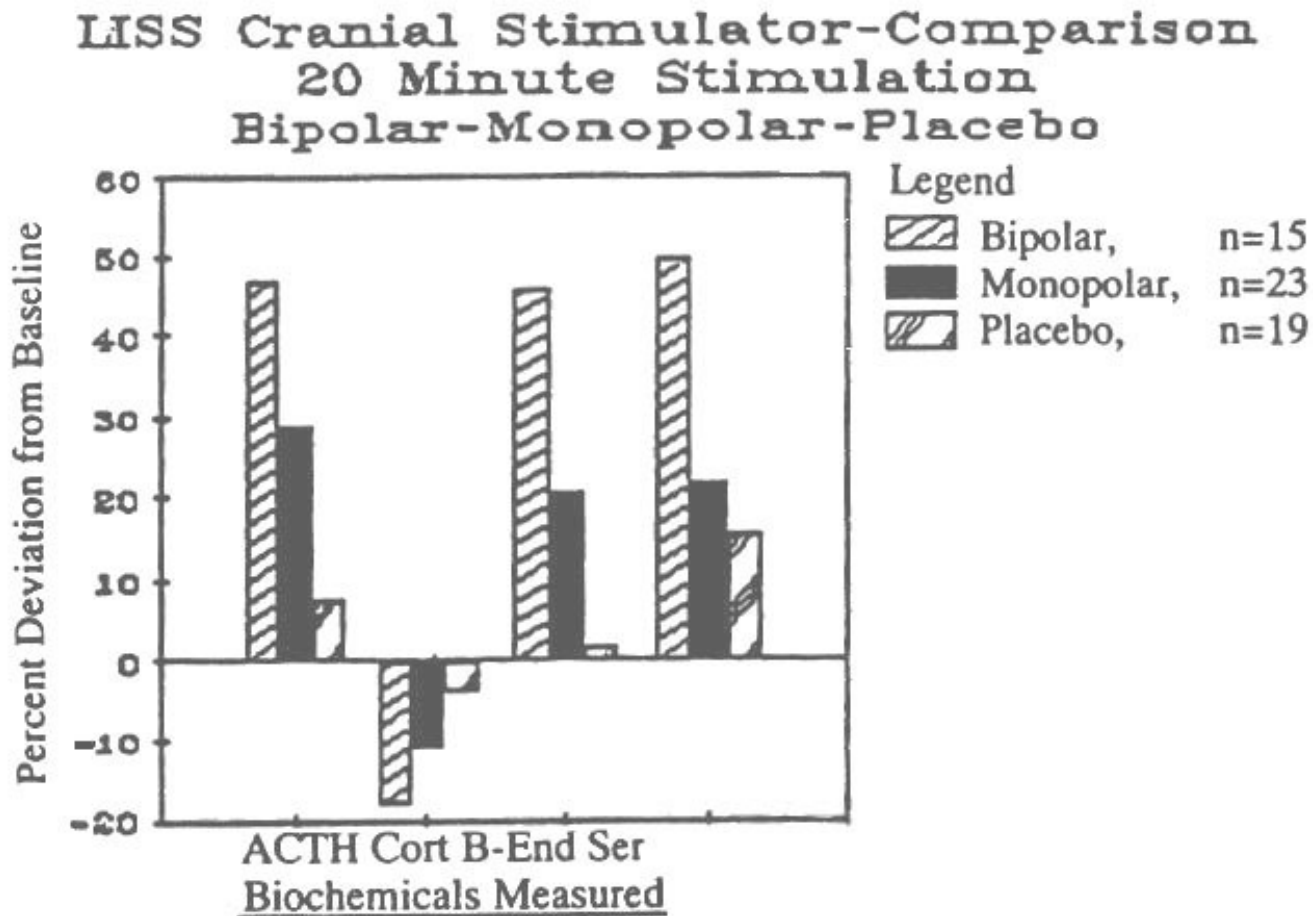


FIG. 1. Biochemical changes following 20-minute transcranial treatment with the LISS Stimulators-monopolar, bipolar and placebo.

**Table 10.2** Steps in sensory transduction

Transduction step	Vision	Olfaction	Taste		Mechanoreception (hair cells)
			Sweet/bitter amino acids	Salt/sour	
Energy	Photons	Molecules	Molecules	$\text{Na}^+$ , $\text{H}^+$	Displacement
Membrane receptor	7TD family: rhodopsin	7TD family: olfactory	7TD family: gustatory		
G protein	Transducin	$G_{\text{olf}}$	$G_{\text{gust}}$		
G-protein target	Phosphodiesterase	Adenylate cyclase III; phospholipase C	AC; PLC		
Second messenger	cGMP	cAMP; $\text{IP}_3$	cAMP; $\text{IP}_3$		
Protein kinase			Protein kinase A?		
Membrane channel	Cationic; inward	Cationic; inward Anionic; inward	$\text{K}^+$	$\text{Na}^+$ ; $\text{K}^+$	Cationic; inward
Sensory response	Close channel	Open channel	Close channel	Open; close	Open channel
Adaptation mechanism	$\text{Ca}^{2+}$ ; phosphorylation?; arrestin	$\text{Ca}^{2+}$ ; protein kinases ?	?	?	Myosin/actin motor; $\text{Ca}^{2+}$ ?
Cell body output	Synapses	Impulses	Synapses	Synapses	Synapses

7TD family: 7 transmembrane domain receptor family.

From Shepherd (1991b).



# Cyclic AMP Response to Electricity

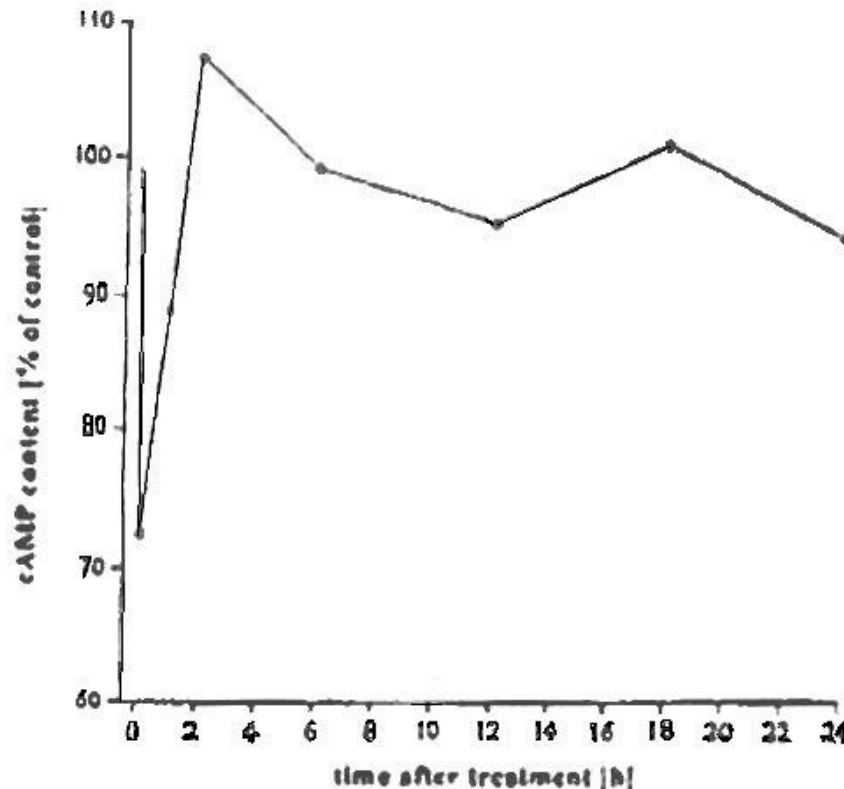
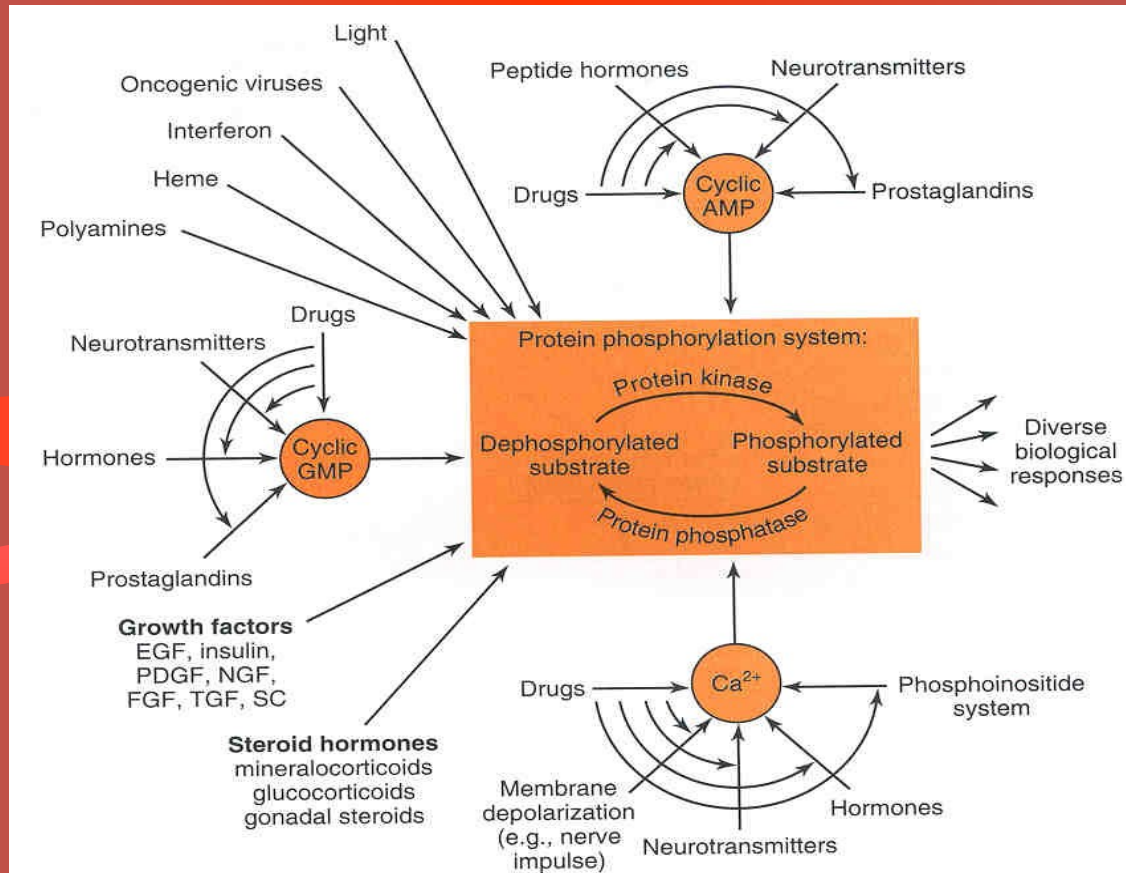


Fig. 2. Cellular cAMP content of SV40-3T3 cells as function of time after treatment with a sine wave electric 4000-Hz field (one experiment). Exposure conditions: 5 min at 1 V/m ( $250 \mu\text{A}/\text{cm}^2$ ). Results are given in percent of control cells (100%) harvested at the same time as the exposed SV40-3T3 cultures.

# The Essence of a Second Messenger



**FIG. 1.** Schematic diagram of the role played by protein phosphorylation in mediating some of the biological effects of a variety of regulatory agents. Many of these agents regulate protein phosphorylation through altering intracellular levels of a second messenger, cyclic AMP, cyclic GMP, or  $\text{Ca}^{2+}$ . Other agents appear to regulate protein phosphorylation through mechanisms that do not involve these second messengers. Most drugs regulate protein phosphorylation by affecting the ability of first messengers to alter second-messenger levels (*curved arrows*). A small number of drugs (e.g., phosphodiesterase inhibitors,  $\text{Ca}^{2+}$  channel blockers, lithium) regulate protein phosphorylation by directly altering second-messenger levels (*straight arrows*). (EGF) epidermal growth factor; (PDGF) platelet-derived growth factor; (NGF) nerve growth factor; (FGF) fibroblast growth factor; (TGF) transforming growth factor; (SC) somatomedin C. (From Nestler and Greengard [1].)

# Serotonin & Cyclic AMP

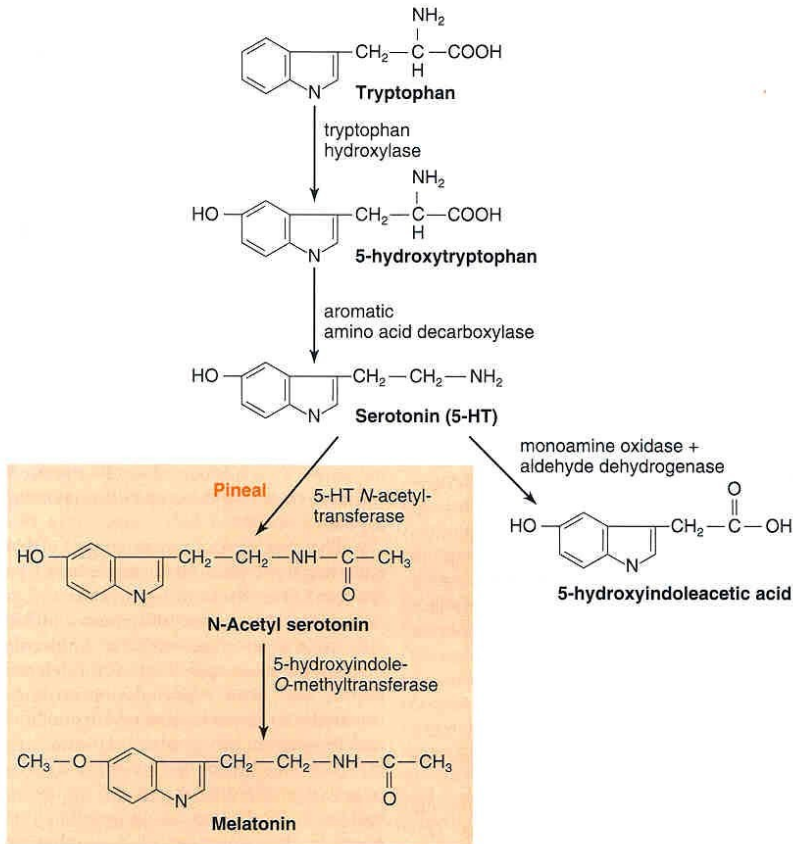
**TABLE 1.** Subtypes of 5-HT receptors in brain

Receptor	Effector mechanism	Clone	Radioligands
5-HT <sub>1</sub>			
1A	cAMP ↓; K <sup>+</sup> channels ↑	+	[ <sup>3</sup> H]DPAT
1B	cAMP ↓	+	[ <sup>125</sup> I]ICYP
1C	PI hydrolysis	+	[ <sup>3</sup> H]mesulergine
1D	cAMP ↓	+	[ <sup>3</sup> H]serotonin
5-HT <sub>2</sub> (D)	PI hydrolysis; K <sup>+</sup> channels ↓	+	[ <sup>3</sup> H]ketanserin
5-HT <sub>3</sub> (M)	Ligand-gated cation channel	+	[ <sup>3</sup> H]zacopride
5-HT <sub>4</sub>	cAMP ↑	—	None

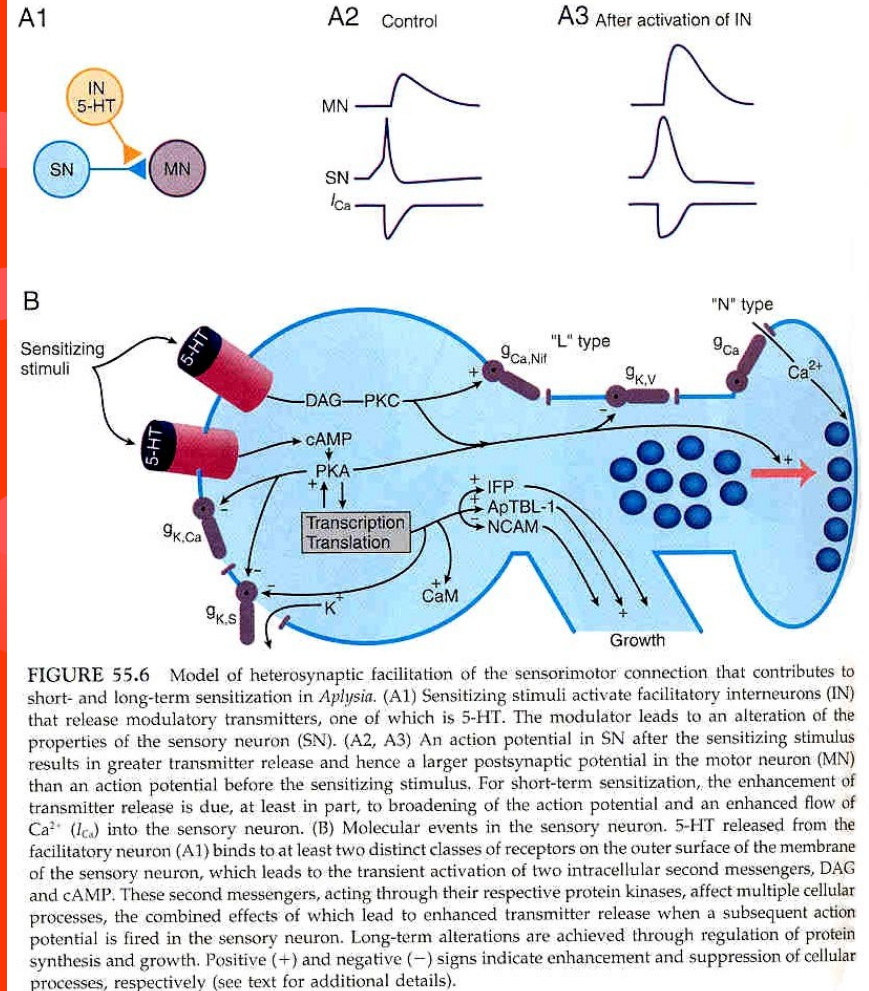


# Serotonin

## PART TWO SYNAPTIC FUNCTION

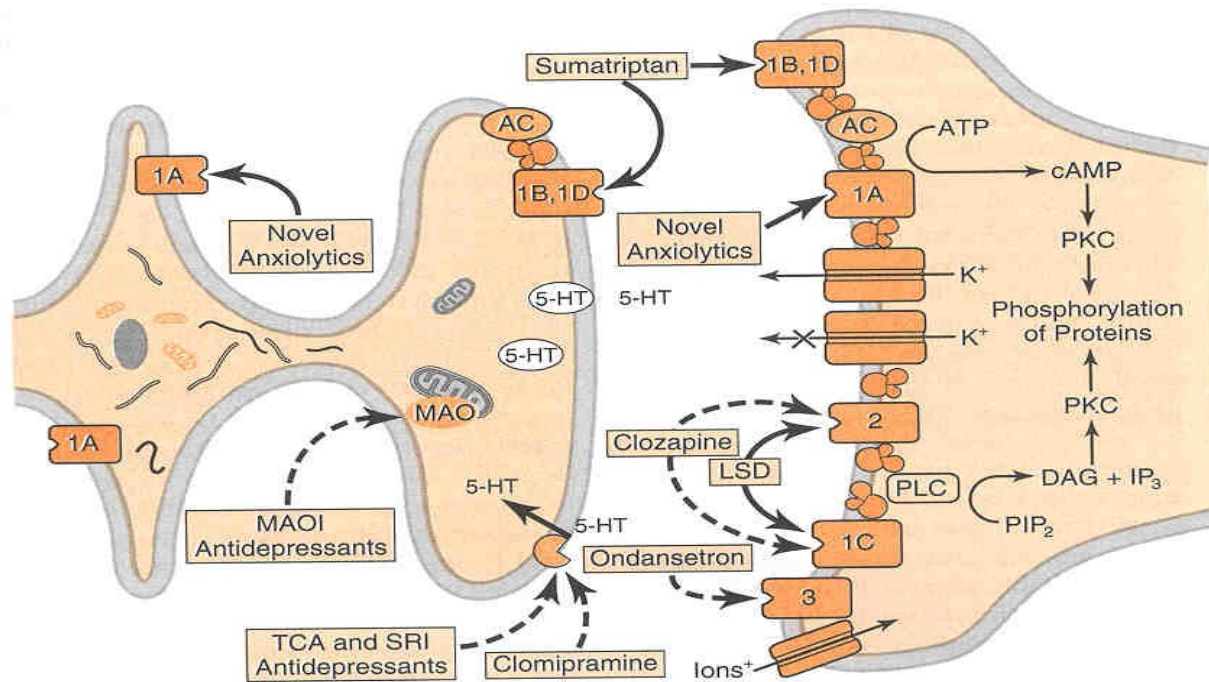


**FIG. 2.** The biosynthesis and catabolism of serotonin. Note that in the pineal gland serotonin is converted enzymatically to melatonin.



**FIGURE 55.6** Model of heterosynaptic facilitation of the sensorimotor connection that contributes to short- and long-term sensitization in *Aplysia*. (A1) Sensitizing stimuli activate facilitatory interneurons (IN) that release modulatory transmitters, one of which is 5-HT. The modulator leads to an alteration of the properties of the sensory neuron (SN). (A2, A3) An action potential in SN after the sensitizing stimulus results in greater transmitter release and hence a larger postsynaptic potential in the motor neuron (MN) than an action potential before the sensitizing stimulus. For short-term sensitization, the enhancement of transmitter release is due, at least in part, to broadening of the action potential and an enhanced flow of Ca<sup>2+</sup> ( $I_{Ca}$ ) into the sensory neuron. (B) Molecular events in the sensory neuron. 5-HT released from the facilitatory neuron (A1) binds to at least two distinct classes of receptors on the outer surface of the membrane of the sensory neuron, which leads to the transient activation of two intracellular second messengers, DAG and cAMP. These second messengers, acting through their respective protein kinases, affect multiple cellular processes, the combined effects of which lead to enhanced transmitter release when a subsequent action potential is fired in the sensory neuron. Long-term alterations are achieved through regulation of protein synthesis and growth. Positive (+) and negative (−) signs indicate enhancement and suppression of cellular processes, respectively (see text for additional details).

# Serotonin Receptor



**FIG. 9.** Effects of psychoactive drugs on serotonergic neurotransmission. Drugs that act as agonists are indicated by **solid-line arrows**, whereas antagonists or inhibitors are shown with **broken-line arrows**. The 5-HT<sub>1A</sub> (1A) receptor acts as both a somatodendritic autoreceptor and a postsynaptic receptor; anxiolytic drugs such as buspirone are agonists at this receptor. In terminal fields, the autoreceptor is either the 5-HT<sub>1B</sub> (1B) or 5-HT<sub>1D</sub> (1D) subtype; these receptors also function as postsynaptic receptors. The antimigraine drug sumatriptan is an agonist at these receptors. There are both structural similarity and similarity in the second-messenger coupling of 5-HT<sub>2</sub> (2) and 5-HT<sub>1C</sub> (1C) receptors. Hallucinogenic drugs such as LSD are agonists at 5-HT<sub>2</sub> and 5-HT<sub>1C</sub> receptors, whereas novel antipsychotic drugs such as clozapine are antagonists. The 5-HT<sub>3</sub> (3) receptor, a ligand-gated ion channel, is blocked by drugs such as ondansetron effective in the treatment of chemotherapy-induced nausea and emesis. Another important target for psychotherapeutic drugs is the serotonin transporter, which is blocked by drugs effective in the treatment of depression or obsessive-compulsive disorder (clomipramine). The enzyme responsible for the catabolism of serotonin, MAO, is inhibited by another class of antidepressants. (AC) adenylyl cyclase; (PKC) protein kinase C; (DAG) diacylglycerol; (IP<sub>3</sub>) inositol trisphosphate; (PIP<sub>2</sub>) phosphatidylinositol biphosphate. (This figure was kindly prepared by Dr. William Clarke, Department of Pharmacology, Mt. Sinai School of Medicine, New York, NY.)

# Measured Consequences of Applied ? EMFs

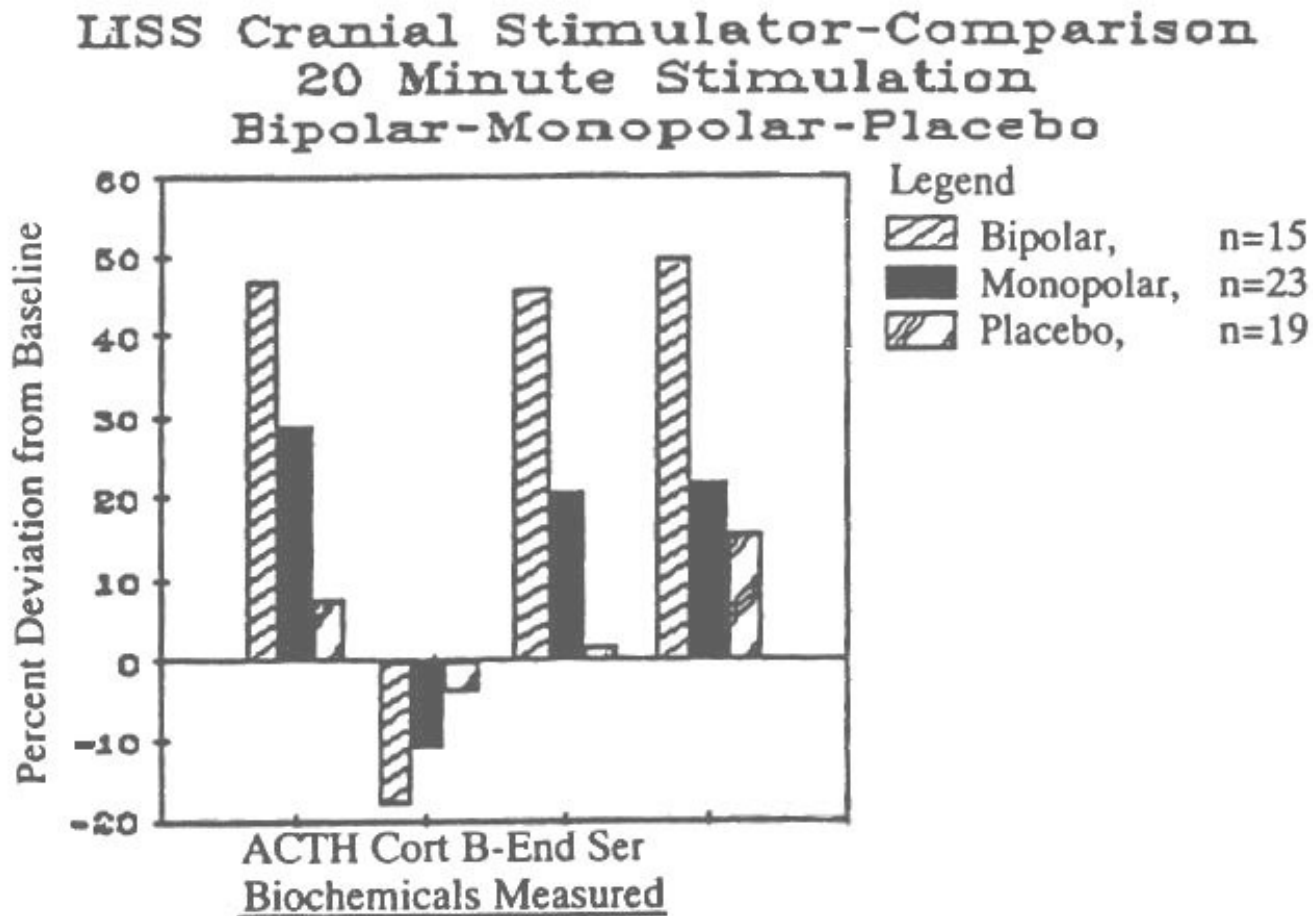


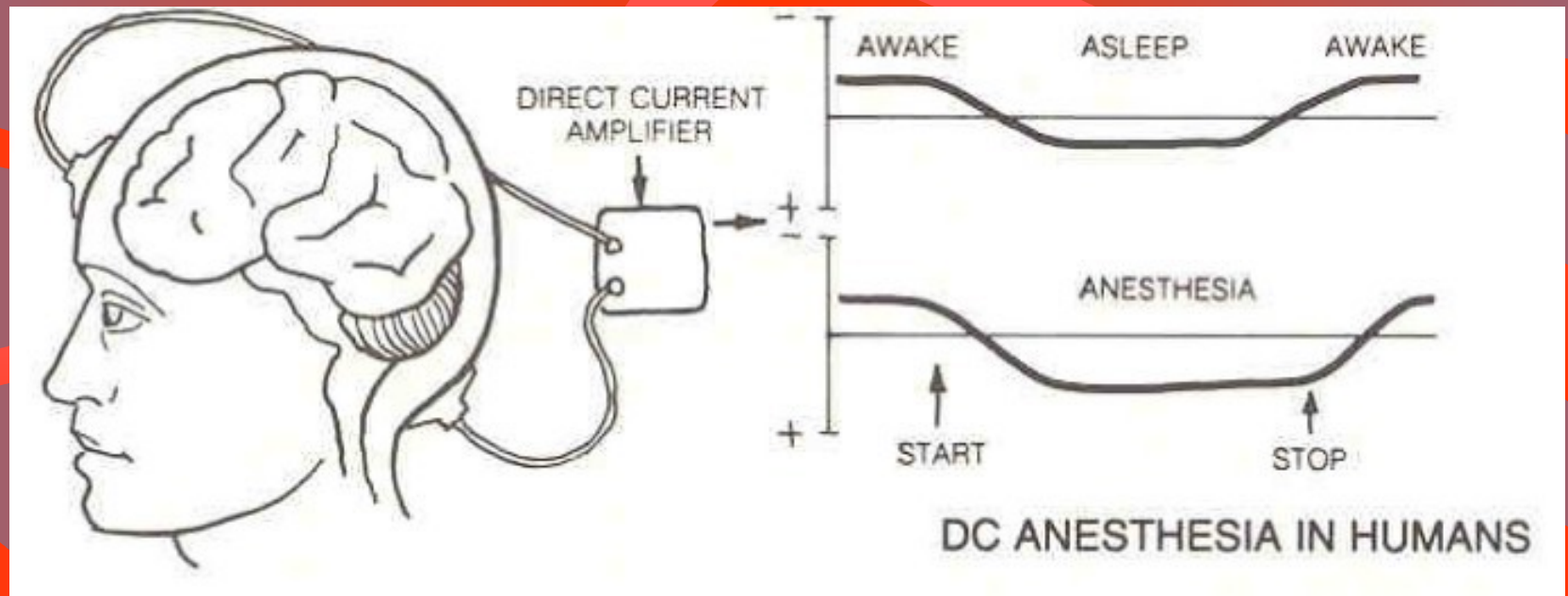
FIG. 1. Biochemical changes following 20-minute transcranial treatment with the LISS Stimulators-monopolar, bipolar and placebo.

# **External Influences**

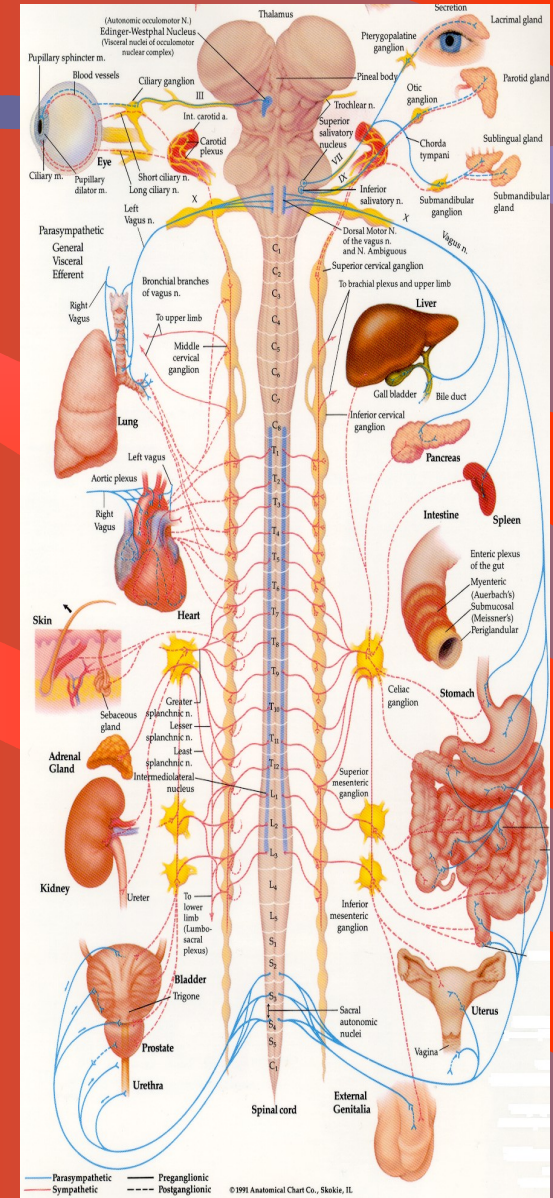
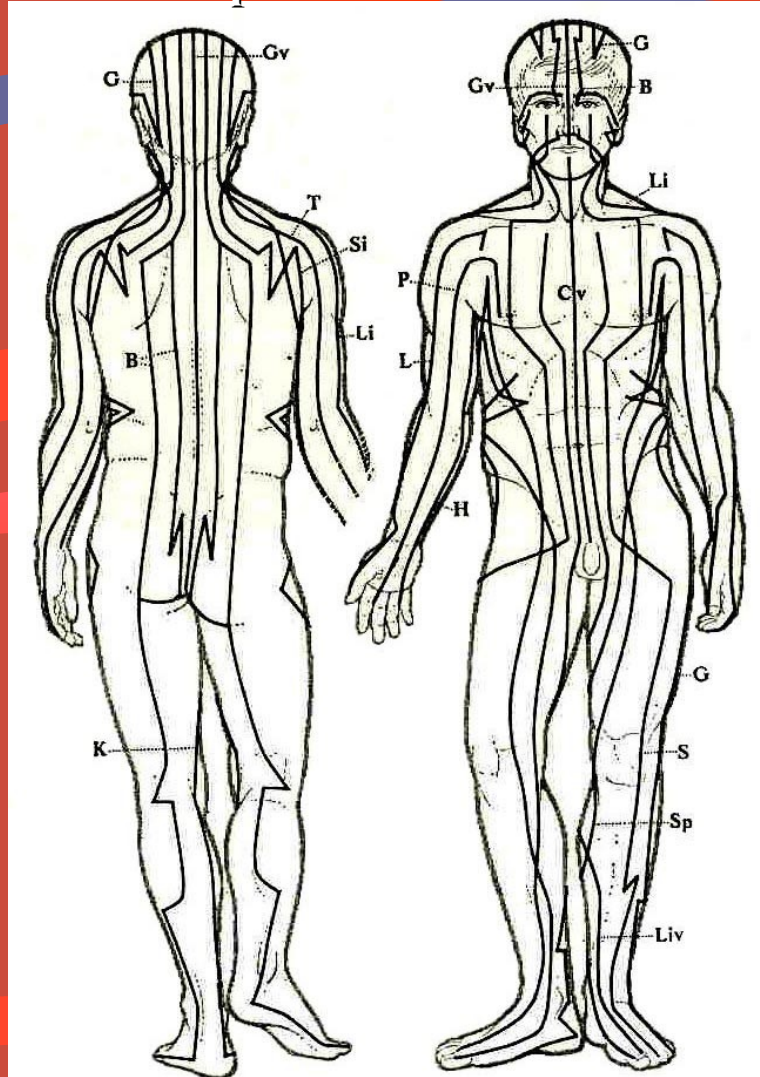
- Magnet Therapy
- Transcranial Stimulation
- Auricular acupuncture
- Electroacupuncture
- Acupuncture
- Reflexology
- Acupressure
- Neural therapy



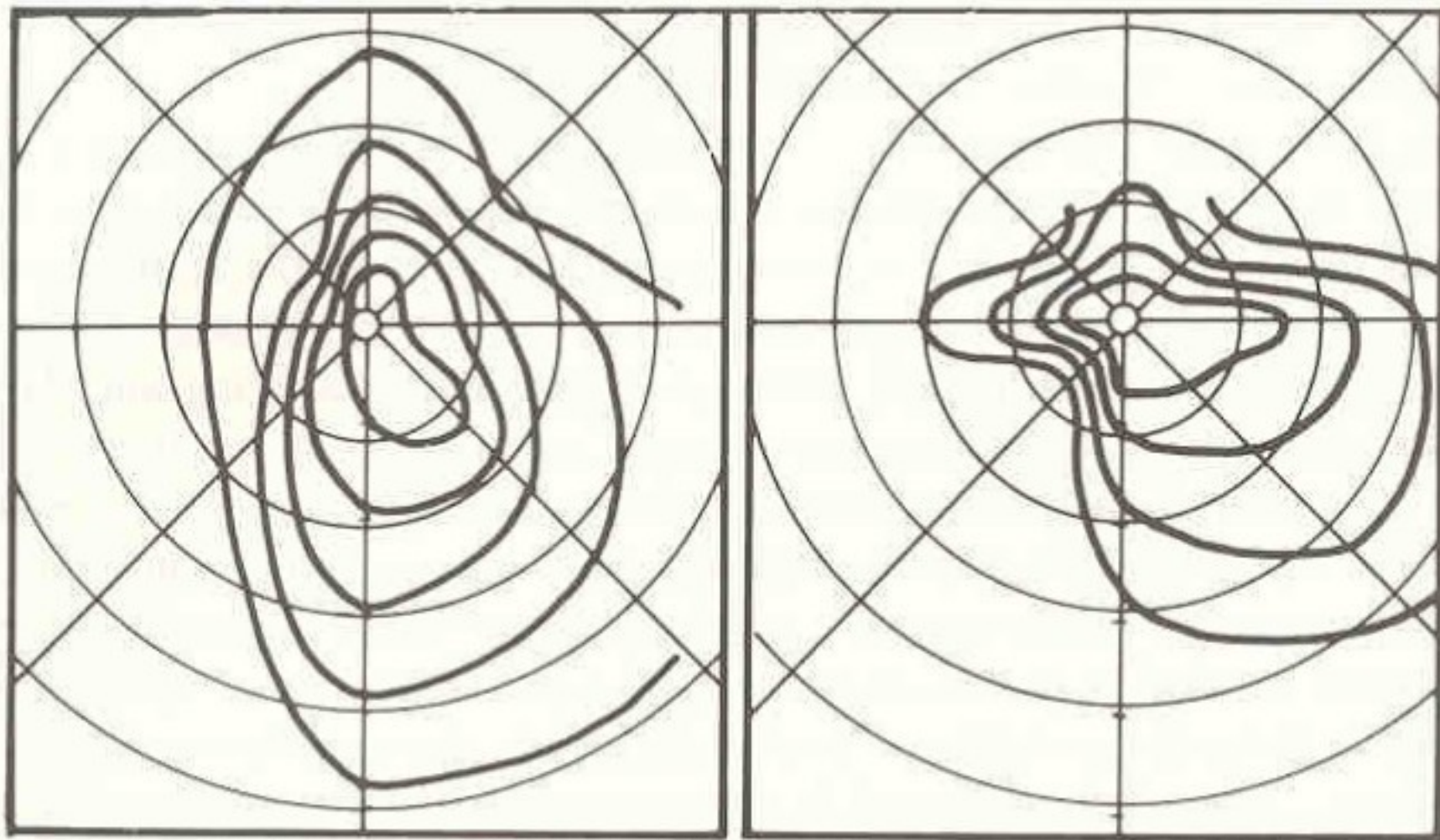
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## Acupuncture Meridians







ELECTRICAL CONDUCTIVITY MAPS OF SKIN AT ACUPUNCTURE POINTS

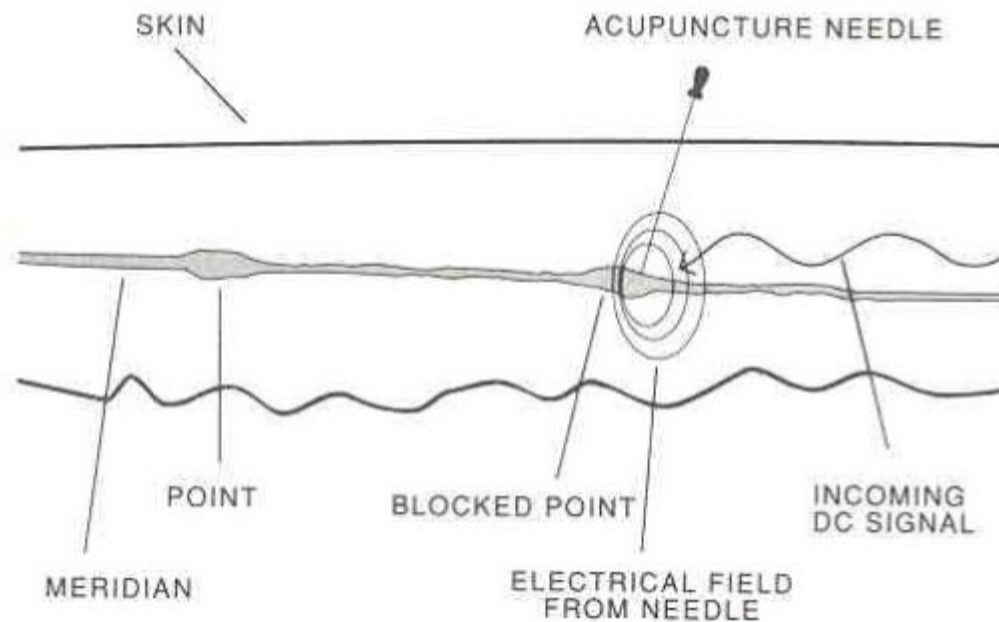


FIGURE 5-1. *A theory of how traditional acupuncture works, as derived from our studies. Since the system would use the minute levels of voltage and current that could be generated by the body, the small electrical field developed by the metallic needle is sufficient to prevent the treated point from amplifying the incoming DC signal and passing it on to the next point. While the points have not yet been anatomically identified, the evidence for their presence is excellent. No effective study has been done to locate the structures associated with the points identified by electrical parameters.*



# Psychosomatic?

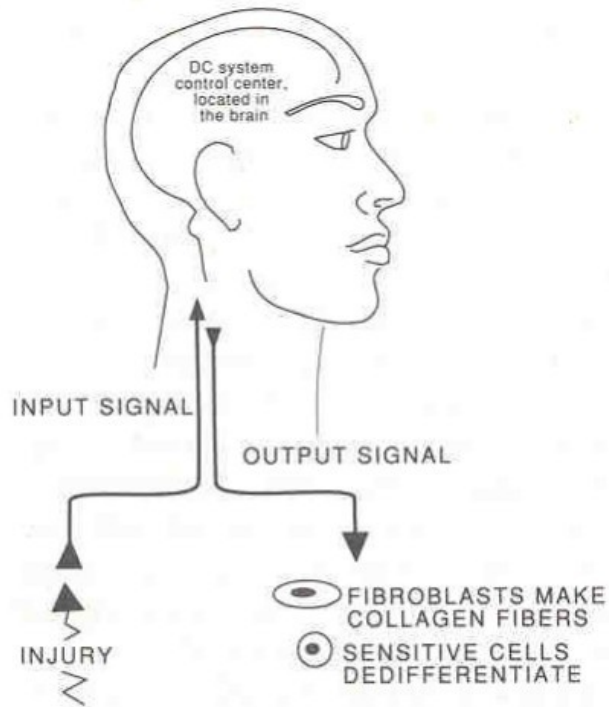


FIGURE 2-7. *The complete closed-loop, negative-feedback growth control system. The input side is the acupuncture system of points and meridians. In the interest of clarity, the “points” that are the booster amplifiers of the acupuncture system are omitted. At this time the nature of the output system was uncertain, except for the fact that it had to be connected with the nervous system as we know it.*

# Conclusions

- True Clinical Results
- Plenty of Science
- Natural Governors = Safe
  - serotonin results
  - maximize with precursors
  - other examples
- Infinite Possibilities
  - Charge Movement
  - Electromagnetic catalyst

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