

RELATIONS and THEMATIC ROLES), case, ANAPHORA, HEAD MOVEMENT, subject distribution, *wh*-questions (WH-MOVEMENT), scrambling, and clitic inventories and placement.

In phonological theory, the shift from serial, process-oriented frameworks (PHONOLOGICAL RULES AND PROCESSES) to OT's parallel, violable constraint optimization has enabled explanation of typological variation in a number of areas: segmental inventories, syllable structure, STRESS, TONE, vowel harmony, reduplicative and templatic MORPHOLOGY, phonology-morphology relations, the phonology-PHONETICS interface, and many others. (For an extensive bibliography and on-line database of OT papers and software, see the Rutgers Optimality Archive ROA at <http://ruccs.rutgers.edu/roa.html>.)

A unified grammatical framework for syntax and phonology, OT also provides results that span both these modules, including the relation of general to more specific constraints, the compatibility among related grammatical processes, and the computation and learnability of grammars. Formal results on the latter topics address algorithms for learning constraint rankings from positive examples, algorithms for computing optimal forms, and the complexity of formal languages specified by OT grammars. Empirical findings on the course of acquisition of PHONOLOGY in children, and on real-time SENTENCE PROCESSING, have been analyzed within OT. While detailed OT proposals for the neural basis of language and the neural basis of phonology do not currently exist, theoretical connections between optimization in OT and in NEURAL NETWORK models have proved fruitful for the continuing development of both OT and the theory of complex symbol processing in neural networks (Prince and Smolensky 1997).

See also CONNECTIONIST APPROACHES TO LANGUAGE; LANGUAGE, NEURAL BASIS OF; PHONOLOGY, NEURAL BASIS OF

—Paul Smolensky

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Origins of Intelligence

See COGNITIVE ARCHAEOLOGY; EVOLUTIONARY PSYCHOLOGY; INTELLIGENCE; MACHIAVELLIAN INTELLIGENCE HYPOTHESIS

PAC Learning

See COMPUTATIONAL LEARNING THEORY; MACHINE LEARNING

Pain

To a degree matched by no other component of somatic sensation and by no other sensory system, pain carries with it an emotional quality. From one person to the next, differences in personal traits and past experience play major roles

in the perception of pain. For any individual, changes in mood or expectation are similarly important for judging and reacting to pain. Moreover, thresholds for a stimulus perceived as painful vary across the body surface, as every person can attest to by comparing how he or she reacts to one grain of sand under the eyelid vs. thousands of grains under the feet. These variations in the perception of pain and its strong affective component make it difficult to study clinically and experimentally.

The anatomy and physiology of pain (nociception) begin with two types of specialized receptors in the skin, muscles, and viscera. One responds only to very forceful mechanical energy and the other to noxious stimuli of many kinds. The first of these is the mechanical nociceptor, a type of afferent that responds only to physical force intense enough to produce tissue damage. Far more general is the response of polymodal nociceptors, as seen by comparing the response of the two receptor types to heat: mechanical nociceptors have a very high threshold for the initial application of heat, whereas polymodal nociceptors begin responding to stimuli of 40° C and show a linear increase in response to stimuli up to 60° C. Both receptor types are notable for the fact that, unlike all other somatosensory receptors, they are left uncovered by specialized cells or connective tissue sheaths and are thus unprotected from the diffusion of chemical agents released by surrounding cells. These agents include a variety of small molecules such as amines and peptides that can produce or change activity in nociceptors over a distance of several millimeters.

Whereas other somatosensory receptors adapt to repeated stimulation by becoming less sensitive and less responsive to each subsequent stimulus, nociceptors participate in a heightened response to repeated noxious stimulation, referred to as hyperalgesia. Both neural and non-neuronal mechanisms appear to participate in this phenomenon by which application of noxious thermal or mechanical stimuli produces a lower threshold for and a greater response to other noxious stimuli. Primary hyperalgesia occurs at the site of injury through the local release of chemical agents, such as bradykinin, which directly stimulate nociceptors to become active. Other chemical agents, including prostaglandin E₂, also play a role in primary hyperalgesia, not by directly driving nociceptors, but by making them much more responsive to subsequent non-noxious mechanical stimuli. It is through inhibition of these chemical agents that aspirin and ibuprofen work as analgesics. A second type of hyperalgesia is of strictly neural origin and probably includes a component that originates in the spinal cord rather than in the periphery.

The two nociceptor types send their responses into the central nervous system (CNS) by way of different kinds of peripheral axons. Larger, lightly myelinated (A δ) axons end as mechanical nociceptors, whereas the smallest, unmyelinated (C) axons end as polymodal nociceptors. These differences in axon diameter and level of myelination necessarily translate into differences of conduction velocity and thus in the time over which signals from the two nociceptor types reach the CNS. Pricking pain, carried by A δ fibers, is the more rapidly transmitted, better localized, and more easily tolerated component of pain. The perception of pricking

pain is followed after a substantial delay by second or burning pain, a poorly localized, agonizing pain carried by C fibers.

Pain afferents are segregated from other somatosensory afferents that carry discriminative information of touch and body position. They enter the spinal cord in the dorsal root as a more lateral bundle and synapse directly upon neurons of the cord's dorsal and intermediate horns. Convergence of inputs from many pain afferents at this level produces a situation amplified at higher levels in which painful stimuli are localized very poorly when unaccompanied by information from other cutaneous afferents. Further synaptic convergence at higher levels between cutaneous nociceptors and visceral nociceptors leads to the misplacement of pain occurring in viscera to sites that are more peripheral. This referred pain is a common part of abnormal situations, such as those that occur during heart attacks.

Many spinal neurons driven by nociceptive afferents send their axons across the spinal cord, where they ascend to various locations in the brain stem and THALAMUS. That immediate crossing in the cord of pain information contrasts with the delayed crossing of fine touch and proprioceptive axons, which occurs in the medulla. Such a wide difference in the site of crossing produces a situation in which hemisection of the cord leads to a loss of pain sensation on the contralateral side of the body but loss of discriminative sensation on the ipsilateral side. Other neurons directly driven by nociceptive afferents have intraspinal axons that end on spinal motor neurons. These synapses are part of a rapid reflex that produces withdrawal of a limb from the location of a painful stimulus.

Much of the ascending pain information reaches a region of the midbrain around the cerebral aqueduct of the ventricular system. This periaqueductal gray (PAG) region is a principal component of a descending system, stimulation of which relieves pain. Axons from the PAG innervate a collection of serotonin-synthesizing and -secreting neurons of the medulla, the nucleus raphe magnus. These neurons, in turn, send long, descending axons into the dorsal horn of the spinal cord, where they form synapses with interneurons that use opiate-like peptides, the enkephalins, as NEUROTRANSMITTERS. By modulating the activity of nociceptor afferents and of spinal pain neurons, the enkephalinergic interneurons control the perception of the intensity of a noxious stimulus. Opiates and other pharmacological agents that mimic the effects of enkephalins are effective as analgesics in part because of their action at these spinal synapses.

A fraction of spinal neurons that respond to nociceptive inputs send their axons to the contralateral thalamus. Part of that spinothalamic system reaches nuclei of the intralaminar group, which provides the great mass of the cerebral cortex with a diffuse innervation. That system and the projection of spinal nociceptive neurons to the brain stem reticular formation are the anatomical substrates for the generally arousing and motivating qualities of pain. Most spinothalamic axons, however, synapse on clusters of small cells in the ventral posterior lateral (VPL) nucleus, which together make up a complete body representation of pain. A comparable group of cells in the ventral posterior medial (VPM) nucleus is innervated by neurons in the pars caudalis of the spinal trigeminal system and includes a nociceptive representation

of the face. Both VPL and VPM send axons to the first somatosensory area of the cerebral cortex, found in the postcentral gyrus of monkeys, apes, and humans. By this route and by a separate innervation of the second somatosensory area, nociceptive information reaches the CEREBRAL CORTEX in a way that it can be compared with other somatosensory information and localized with some precision to particular sites along the body. Neurological studies of soldiers suffering head wounds in the two world wars clearly demonstrate that injuries confined to the postcentral gyrus produce permanent analgesia along the contralateral body surface.

Where the affective quality to pain arises is poorly understood. Those few studies to have addressed the question have focused on areas of temporal and orbitofrontal cortex in humans and nonhuman primates. Perhaps the best current guess is that more than one area of cerebral cortex is involved in the agony that accompanies extreme pain such as that produced by solid tumors or burns. These extreme cases of pain and the need to control them and other lesser or more acute nociceptive events often raise questions of the advantage conferred by painful affect. Rare clinical cases of patients who perceive a painful event as differing from an innocuous stimulus but who experience no affect accompanying that event are test cases for such a question. Most of these patients die at an early age, victims of numerous destructive wounds and crippling conditions of joints. Apparently the failure of these patients to avoid or discontinue actions that are painful significantly shortens their lives despite intensive training in detecting and responding to painful stimuli. From these cases, then, it can be concluded that both precise localization and emotional reaction to pain are parts of successful strategies for survival.

See also EMOTION AND THE ANIMAL BRAIN; EPIPHENOMENALISM; PHANTOM LIMB; WHAT-IT'S-LIKE

— Stewart Hendry

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Parallelism in AI

See COGNITIVE MODELING, CONNECTIONIST; NEURAL NETWORKS

Parameter-Setting Approaches to Acquisition, Creolization, and Diachrony

How is knowledge of one's idiolect—*I(nternal)-language*, in Noam Chomsky's (1986) terminology—represented in the mind/brain? How is such knowledge acquired by children? Answers to these questions are intricately and constructively related. In the principles and parameters/minimalist approach (Chomsky 1981, 1986, 1995; see SYNTAX, ACQUISITION OF and MINIMALISM), linguistic knowledge, in addition to a (language-specific) LEXICON (see WORD MEANING, ACQUISITION OF and COMPUTATIONAL LEXICONS), consists of a computational system that is subject to an innate set of formal constraints, partitioned into *principles* and *parameters*. The *principles* are argued to be universal; they formalize constraints obeyed by *all* languages (see LINGUISTIC UNIVERSALS). Alongside these principles—and perhaps *within* some of these principles (e.g., Rizzi 1982)—what allows for diversity in TYPOLOGY (possibly, in addition to the lexicon proper) are the *parameters*. These parameters constitute an innate and finite set of “switches,” each with a fixed range of settings. These switches give the learner a restricted number of options in determining the complete shape of the attained I-language. In such a framework, syntax acquisition reduces to fixing the values of parameters on the basis of primary linguistic data (PLD) (cf. LANGUAGE ACQUISITION). Taken together, principles and parameters bring a solution to the “logical problem of language acquisition”:

$$\begin{aligned}
 (1) \quad & \text{UG} / S_0 \\
 & \text{(universal principles cum UNSET parameters)} \\
 & + \\
 & \text{PLD} / \text{“triggers”} \\
 & = \\
 & \text{Idiolect-Specific Grammar} / S_f \\
 & \text{(universal principles cum SET parameters)}
 \end{aligned}$$

Per (1), language acquisition is the process in which exposure to PLD transforms our innately specified *faculté de langage* (from an initial state S_0) into a language-specific grammar (at the final state S_f) by assigning values (settings) to an array of (initially unset) parameters (see Chomsky 1981, HISTORICAL LINGUISTICS, and INNATENESS OF LANGUAGE).

The schema just sketched delineates a fascinating and productive research program. Yet our understanding is still very incomplete as to how (which aspects of) the PLD “lead” the learner to adopt (what) settings for (what) parameters. What are the major questions raised by this program? In order to flesh out the structure in (1), generativists are advancing on three complementary theoretical fronts toward:

1. A characterization of parameters. For example, are parameters distributed across various grammatical principles (cf. Rizzi 1982; Chomsky 1986) or are parameters restricted to “inflectional systems” (Borer 1983; Chomsky 1995: ch 2), to the inventory and properties of functional heads (Ouhalla 1991; cf. SYNTAX and HEAD