COMPUTER MODELING IN PSYCHOLOGY

Chaired by Frederick J. Bremner, *Trinity University*

An object-oriented simulation of a brain system underlying human aggression

MARK HARTLEY and DAVID V. REYNOLDS
University of Windsor, Windsor, Ontario, Canada

Neural structures involved in aggression were incorporated into a model on the basis of neuro-anatomical data. A software program written in Smalltalk, the Brain Structure Simulator (Seguin, 1994) was used to simulate the effects of activity levels of different brain structures. Relationships among brain structures were then tested by changing activity levels of key structures. Results were consistent with research reports on the brain basis of aggression (Albert, Walsh, & Jonik, 1993; Pontius, 1984).

There is historical precedent for hypothesizing large-scale brain systems that mediate emotion (Papez, 1937), but detailed neural systems for emotional behavior such as aggression have seldom been proposed. One reason may be the lack of a practical method for evaluating brain systems of aggression.

The development of object-oriented and other programming strategies, however, has made computer simulations of brain systems practical (Petherick, Reynolds, Seguin, & Mikes, 1995). Computational models can simulate brain structure interaction and provide a way to determine whether a hypothesized system will function in a way that is consistent with empirical research. The present study used an object-oriented strategy to simulate a neural system of aggression.

Neurologically, human aggression may most closely parallel defensive animal aggression, since brain regions in humans where stimulation or lesions alter aggression are essentially the same as the neural system mediating defensive aggression in animals (Albert, Walsh, & Jonik, 1993).

A computer simulation depends on an explicit representation of the system to be modeled (Whicker & Sigelman, 1991). For the study described here, a brain diagram was developed that was based on neural structures reported to be involved in human aggression, and excitatory and inhibitory projections were incorporated into this representation. A subset of eight structures and their excitatory and inhibitory connections were then entered into the simulation software in the following order: amygdala (AMY), hypothalamus (HYP), periaqueductal gray (PAG), ventral tegmental area (VTA), septal area (SA),

orbitofrontal cortex (OFC), inferior temporal cortex (ITC), and cerebellum (CER) (see Figure 1).

Tests were then conducted to determine whether the system could function normally. During a simulation, when the value of one structure is changed, the values of the other structures are altered and changes are passed through the system. For a system to function normally, changes passing through the system will tend to cancel each other out over a number of cycles. Stability testing involved changing the activity levels of simulated structures to model normal fluctuations in brain activity. After the simulation showed it could perform with reasonable stability, simulation experiments were conducted by changing the activity levels of key brain structures.

METHOD

Procedure

A computer program written in the object-oriented programming language Smalltalk was used to simulate dynamic interactions within a network of brain structures thought to underlie human aggression. The software, Brain Structure Simulator (BSS; Seguin, 1994), runs from diskette or hard disk on PC-type microcomputers. To simulate a neural system, the names of structures and their connections were entered by keyboard. The program initially assigns a value of 100 to each structure to represent normal functioning.

Using a series of mouse-activated menus, different structures are assigned different activity levels and the simulation of brain structure interaction is initiated. During simulations, the value of a source structure is added or subtracted to the value of a destination structure receiving a projection. For excitatory connections, the source structure amount above 100 is added and the amount below 100 is subtracted.

The rules for inhibition are the same, except that values greater than 100 are subtracted from the receiving structure and values less than 100 are added. Whenever both sending and receiving structures have a value of 100, their activity levels do not change. The sequence in which one structure affects another makes a difference

Correspondence should be addressed to D. V. Reynolds, Department of Psychology, University of Windsor, Windsor, ON, Canada N9B 3P4.

Figure 1. Brain structures incorporated into a computer model of a neural system for human aggression. Numbers indicate the sequence of interactions among nuclei during simulation. For brain structures with two-way projections, the connections on the right or above are executed first. A, amygdala; CER, cerebellum; H, hypothalamus; ITC, inferior temporal cortex; OFC, orbitofrontal cortex; PAG, periaqueductal gray; SA, septal area; VTA, ventral tegmental area.

in the simulation and is determined by the order in which connections are entered in the simulator program. Activity levels of the structures typically range from 0 to 200, with 100 considered normal.

A model of human aggression based on published research was developed to identify the brain structures involved and demonstrate the hypothesized excitatory or inhibitory effect of their interaction. This model was simplified (Figure 1) and then run on the simulation software. Simulation A was developed to determine whether the system could mediate an immediate aggressive response. Since AMY stimulation has been shown to trigger outbursts of aggression in both animals and humans (Albert et al., 1993), the activity level of this structure was increased by an arbitrary amount while all other values were at 100. The effect of increasing AMY to 105 is shown (initial change in Figure 2). Simulation B was designed to determine whether the OFC, which is involved in mediating the suitability of an aggressive response to different stimuli (Pontius, 1984), could modify an aggressive response. A simulation for a normal aggressive response was run for two cycles and then the value of the OFC was also increased from 100 to 105. At the end of six cycles, the OFC was reduced from 105 to 100, and the simulation continued for a number of cycles. Subtracting from the OFC in Cycle 6 represented this structure's return to normal after modifying the AMY response.

RESULTS

After trial-and-error alterations in the pattern of connectivity among simulated brain structures and the incorporation of an additional brain structure to improve the model (CER; Daum & Ackerman, 1995), the computer model achieved stability. In Simulation A, designed to determine whether the system could simulate an immediate aggressive response, the AMY was increased to 105 to reflect an environmental activation of the system. As shown in Figure 2, after one cycle of interaction, the AMY returned to 100 (normal), the activity level of the PAG became 95, and the CER increased to 105. At the end of the second cycle, the AMY was at 95 and all other activity levels were at 100. In Cycle 3, the PAG changed from 100 to 105 and the CER fell from 100 to 95. Cycle 4 activity levels for all brain structures were identical to those in the initial cycle.

During Simulation B, designed to test the ability of the OFC to modulate an ongoing aggressive response, the

Figure 2. Activity levels of brain structures in a simulated aggressive response to threatening stimuli. AMY, amygdala; CER, cerebellum; HYP, hypothalamus; ITC, inferior temporal cortex; OFC, orbitofrontal cortex; PAG, periaqueductal gray; SEP, septal area; VTA, ventral tegmental area.

activity levels of all brain structures except the AMY and CER returned to 100 in the seventh cycle. Figure 3 shows the changes in activity levels of the AMY, CER, and PAG. The activity levels found in the seventh cycle subsequently repeated every four cycles. For Cycles 3–6 (after the OFC was increased at the end of the second cycle and before the OFC was decreased after Cycle 6), the values of the AMY changed from normal to above and back (100, 115, 105, 100), the CER changed from below normal to normal and back (85, 95, 100, 95), and PAG increased in Cycle 3, decreased successively in Cycles 4 and 5, and began to increase in Cycle 6 (105, 100, 85, 95). In Cycles 7–10, after the OFC was reduced at the

end of Cycle 6 (again, see Figure 3), the PAG began to go down and up every four cycles (100, 95, 90, 95) so that its activity level stayed between 90 and 100. Also in Cycles 7–10, the AMY rose and fell (105, 110, 105, 100), and the CER also fluctuated but with different values (90, 95, 100, 95). Cycles 7–10 repeated continually.

DISCUSSION

In the “real” brain, aggressive behavior is mediated by a number of brain structures including, but not limited to, the amygdala, hypothalamus, periaqueductal gray, ventral tegmental area, septal area, inferior temporal cor-

Figure 3. Activity levels of brain structures in a simulation of orbitofrontal cortex intervention during an ongoing aggressive response. AMY, amygdala; PAG, periaqueductal gray; CER, cerebellum.

tex, orbitofrontal cortex, and cerebellum. This aggression may take the form of an immediate reaction and can be modulated depending on situational demands. The computational model's ability to mediate both primary and modulated aggressive responses was demonstrated in the two simulations. In the test for a primary response (Simulation A), the PAG quickly became active whereas the CER became proportionately inactive. This pattern emerged after three cycles, with the entire system returning to its starting point in four cycles (Figure 2). The model's capacity to modify a simulated aggressive response

was demonstrated in Simulation B (Figure 3) when the influence of the OFC prevented the PAG from rising above 100 after the third cycle.

The brain embodies neural structures that either excite or inhibit other brain structures (Farah, 1994). The software used in this study simulates a neural system by representing brain structures as input/output units that follow a simple set of rules to change the activity of other brain structures. To show normal function, both the real brain and the computer model must exchange excitatory and inhibitory messages in a way that permits the overall

system to remain in a state of equilibrium. Abnormal function is shown when the activity levels of structures in the system take on extreme values or fluctuate erratically.

The simulations used in this study exploit a conceptual approach that was developed outside cognitive neuroscience. The approach, called object-oriented programming, was intended to make computer software development more efficient (Booch, 1991), but it also embodies insights for both simulating and understanding brain function. The underlying structure of object-oriented software has aspects in common with the dynamic, modular architecture of the mature human brain. Computational objects receive, process, and send messages to other computational objects (Entsminger, 1990). Similarly, brain structures receive selective input from other brain structures, respond to this input by changing activity, and then project selectively to other brain structures. In short, brain structures can be conceptualized as "objects" in an object-oriented simulation (Reynolds, Getty, & Atwell, 1995).

Interestingly, in spite of the simplicity of the system shown in Figure 1, feedback among the structures resulted in nonlinear changes in the different brain structures following successive cycles of the simulation (see Figure 3). This emergent nonlinearity, which is a characteristic of brain activity (Crick, 1995), was achieved despite the linear add/subtract algorithm between adjacent structures.

Finally, the potential utility of the simulation is not limited to understanding the way in which brain dysfunction may contribute to human aggression. Any complex sys-

tem, conceptualized as interacting modular entities, can exploit the object-oriented strategy reported here.

REFERENCES

- ALBERT, D. J., WALSH, M. L., & JONIK, R. H. (1993). Aggression in humans: What is its biological foundation? *Neuroscience & Biobehavioral Reviews*, *17*, 405-425.
- BOOCH, G. (1991). *Object-oriented design with applications*. Redwood City, CA: Benjamin/Cummings.
- CRICK, F. (1995). *The astonishing hypothesis*. London: Touchstone.
- DAUM, I., & ACKERMAN, H. (1995). Cerebellar contributions to cognition. *Behavioural Brain Research*, *67*, 201-210.
- ENTSMINGER, G. (1990). *The Tao of objects*. Redwood City, CA: M & T Books.
- FARAH, M. J. (1994). Neuropsychological inference with an interactive brain: A critique of the "locality" assumption. *Behavioral & Brain Sciences*, *15*, 285-320.
- PAPEZ, J. W. (1937). A proposed mechanism of emotion. *Archives of Neurology & Psychiatry*, *38*, 725-745.
- PETHERICK, M. A., REYNOLDS, D. V., SEGUIN, M., & MIKES, K. (1995). An object-oriented simulation of anorexia based on brain structure interaction. *Behavior Research Methods, Instruments, & Computers*, *27*, 120-124.
- PONTIUS, A. A. (1984). Specific stimulus-evoked violent action in psychotic trigger reaction: A seizure-like imbalance between frontal lobe and limbic systems? *Perceptual & Motor Skills*, *59*, 299-333.
- REYNOLDS, D. V., GETTY, H. A., & ATWELL, G. (1995). *Object-oriented computer models of brain disorders based on functional neuroanatomy*. Paper presented at the Workshop on Neural Modeling of Cognitive and Brain Disorders, University of Maryland, College Park.
- SEGUIN, M. (1994). *Brain Structure Simulator* [Computer software]. Windsor, ON, Canada: Author.
- WHICKER, M. L., & SIGELMAN, L. (1991). *Computer simulation applications*. Newbury Park, CA: Sage.

(Manuscript received September 30, 1996;
revision accepted for publication January 31, 1997.)