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EDITORIAL

Revamping neurorehabilitation in Oman

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إعادة بناء التأهيل النفس - عصبي في عُمان

سمير بن حمد العدوي و ديفيد برك

A FALLOUT OF THE RECENT AFFLUENCE IN OMAN—and the Gulf region in general—is the sharp rise in traffic accidents and the consequent head injuries, which render many young people to severe morbidity and mortality.[1] Head injuries result in 10% of global mortality and are responsible for more years of life lost than most diseases or medical conditions.[2,3] Though Oman is a world leader in health care, facilities for rehabilitation of the brain injured remain minimal.[4–6] Until recently this was understandable and perhaps excusable, since experts previously considered it impossible to regenerate or anatomically reorganize the adult nervous system, once damaged.[7] This may no longer be the case. Research in the nineties—the decade of the brain—has brought new insights into the pathophysiology, impairments, functional limitations and disabilities involved in brain injury. These may provide a better understanding of the mechanisms of functional recovery following brain damage, and have kindled hopes for more effective rehabilitation.[7,8] It is instructive to review some of the pertinent medical history and then the recent medical progress in our understanding of the response of the brain to injury.

The belief in irreversibility of neural damage probably goes back to the Spanish anatomist and Nobel Laureate, Santiago Ramony Cajal. In his 1928 book DEGENERATION AND REGENERATION OF THE NERVOUS SYSTEM he declared, 'Once development is completed, the sources of growth and regeneration of axons and dendrites are irrevocably lost. In the adult brain, nervous pathways are fixed and immutable; everything may die, nothing is regenerated' (p 750).[9] For decades, his influential words kept health planners in inertia under the impression that it was pointless to try to develop training or rehabilitation programmes to promote something that could not happen.

Cajal's pessimistic views were seriously challenged only in the last two decades. We now know not only that there is continuous growth of brain tissue, but have also begun to recognize that the brain, when injured, begins a process of healing which involves intrinsic growth, reorganization, adaptation and environmental interaction that reverses many of the pessimistic connotations of Cajal's lament.

The understanding of the mechanisms of plasticity was an important development.[10] Plasticity refers not only to possible cortical changes after injuries but also to a wide variety of biological processes relevant to rehabilitation.[11] Among the models and mechanisms suggested to explain plasticity is *vicarience*, which infers that different areas of the brain have the potential to take over specific functions of damaged tissues. The parallel term in modern neuropsychology is equipotentiality, the ability of healthy nerve cells to take over the functions of their damaged counterparts.

Vicarience has been supported by many experiments. Among these are the longitudinal studies on monkeys and rats by Merzenich et al and more recently, by Al-Adawi.[12–13] Following median nerve transection and ligation, the researchers completely deafferented the animals' limbs, thus severing all sensory nerves. Subsequently, 'new' inputs formed in the brain's receptive field of deafferented limbs, suggesting that the brain possesses an inherent capacity for reorganization, leading to functional recovery and possibly, rehabilitation. Similar reorganization has also been reported in humans. Mogilner showed somatotopic reorganization in two adult subjects who were scanned using magnetoencephalography before and after surgical separation of syndactyly (webbed fingers).[14] The presurgical maps had displayed shrunken and nonsomatotopic hand representation. Within weeks of surgery, however, cortical reorganization occurred up to 39 mm from the

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site of the original area of representation, reflecting the new functional status of the separated digits. There are more experimental and clinical reports that reaffirm the mechanism of vicarience.[15] Interestingly, neuropsychological plasticity, rather than being limited by certain critical periods of development, can occur at any age.[15]

The second popular concept of how recovery might occur is based on the notion that the brain evolves 'backup' or 'fail-safe systems' to be activated when something goes wrong. This is similar to backup computers or second brake systems in vehicles, where the standby facility automatically assumes the functions of the damaged system. In neuropsychology, such compensatory mechanism is termed *redundancy*. In the 1970s, Wall and his colleagues demonstrated redundancy by showing that previously silent fibre pathways in the brain stem could become immediately active when the primary sensory fibres in the spinal cord were cut.[16] Since the appearance of activity occurred soon after the injury, Wall proposed that the new pathways were there all the time, but had been masked or inhibited by the active primary sensory fibres. Redundancy (or unmasking) in the nervous system is often used to explain how a patient can retain function after suffering major trauma to the brain.[17]

Such recoveries could also be explained as being due to functional substitution, where part of the brain is considered 'reprogrammed' to take charge of the functions of the damaged area. Recovery is explained not so much by the restoration of normal behaviour as by the development of alternative behaviour that permit patients to achieve certain goals in everyday life, though not as efficiently as before.[18] Examples of functional substitution are demonstrated by individuals such as physicist Stephen Hawking who lost his ability to speak but learned to communicate by using a keyboard. In this conceptual model of rehabilitation, behavioural substitution is expected to be accompanied by structural reorganization of the nervous system itself.[19,20]

The third concept that has received renewed attention is *diaschisis*, which postulates that when injury or disease has disturbed a part, the resultant trauma can affect other quite distant parts. Diaschisis is thought to be a temporary block of function produced by damage or irritation to brain tissue.[21] Recent preliminary studies suggest that after injury, areas of the brain distant from the actual damage site could become depressed.[7] These studies provide evidence that focal subcortical lesions can result in cortical dysfunction in patients even though the cortex is intact, and vice versa. This may explain why non-specific brain injuries sometimes lead to impairments.

In addition to these suggested mechanisms of injury and recovery, another possible mechanism of injury at neurochemical level has begun to emerge.[22] Experiments over the past decades indicate that when a healthy brain is suddenly injured, neurotransmitter activity tends to change.[23] There is consensus that at the earliest phase of a lesion, the injured, traumatized and dying cells become unable to control their fluid and ion balance, and release their stores of amino acid neurotransmitters and calcium ions.[24] The consequent excitotoxicity weakens and 'burns out' the neurons, which eventually die.[19] It is through the understanding of these processes that we now realize that there is a cascade of chemical processes that must occur for a brain cell to die. The understanding of these processes has given us a realization that there may be a number of opportunities to affect the process by which cells die, and perhaps by doing so affect the magnitude of the injury itself. While some of the early studies suggest that there may be a limited window of opportunity during which we may affect these processes, this remains a very fertile area for research and a potentially significant means by which we may reduce the morbidity of our patients.

Each of the areas outlined suggest a response by a medicinal/rehabilitation team that, if not pursued, represent a failure of our medical system to apply the knowledge gained over the past decade to the lives of the citizens who depend on our vigilance. Each advance in knowledge invites more than our passive observation. First, as we discuss the concept of vicarience, we must not view this as an internal process separate from environmental influences. Biernaskie & Corbett have noted that the process of reorganization seems to be heavily influenced by the stresses on the organism as it attempts to engage the damaged neural tissue. [25] For instance, if the damage in the brain results in paralysis of the left arm, it seems the attempt to use the left arm stimulates the organism to more fully reorganize this area of the brain. Without this stress, the reorganization will be less complete. Efforts in rehabilitation elsewhere in the world, which have addressed this, have focused on the forced use of the impaired portion of the brain. There are entire programs that focus on this forced use concept, which are unique to the process of rehabilitation.

The second concept discussed suggests that a redundancy of the brain allows for the 'awakening' of area of the brain when other primary areas have been injured. A point that may be lost in these relatively recent discoveries is that once these areas have been brought "on line" and thus are available for a new function, there is need for this tissue to be trained. If we step back and review the development of the primary tissue, it is clear that there are years of train-

ing during which this tissue originally obtained its facility of function. It is not surprising therefore that there needs to be a process of focused 'rehabilitation' of this new tissue that allows us to maximize its function.

Indeed, over the last few years, more cases of functional recovery after cerebral injury and disease have been published than anytime before.[26] The pathways of neurotransmitters involved have been well charted and the safety of the drugs which influence these transmitters has been relatively well established.[12] It is important to note that although these drugs have helped to tease out neurochemical and pathophysiology behind neurocognitive impairment following brain damage, the traditional role of retraining and remediation (in other words, rehabilitation) plays a central role. In a way, the drug appears to kick-start the system so the retraining or remediation can occur in tandem.[21, 27] We now know that if an area of the brain has been injured that there is a change in the neurochemistry that affects the function of that tissue. If we study the involved neurochemistry, a number of medications are suggested that can introduce or enhance the chemicals that have been altered. With severe injury, the focus of the rehabilitation may be to enhance the ability to attend to the environment. Of the medications most often employed in rehabilitation, neurostimulants are often mentioned prominently. These allow the individual to begin or to maximize his ability to focus on the environment and on the process of rehabilitation. With better focus and longer attention to the learning process the elements of reorganization and teaching of the reorganized tissue can be maximized. Theses are not independent issues. One must be attended while the other is ongoing. This illustrates the new, complex process of neurorehabilaitation.

How practical are the above behavioural and neurochemical descriptions of the mechanisms of injury and recovery? They do suggest that the brain can heal, or be induced to heal, after injury. The individual then can regain self-sufficiency and self worth while he or she is eased into independent living.

If one ponders on the practice and services of rehabilitation in Oman, a sense of helplessness would likely take hold, for the field and practice have been largely disregarded, and worse, there is no such endeavour on the horizon. Each year, 0.3–0.4% of the population of Oman is estimated to incur brain injury, principally from road traffic accidents, domestic accidents and falls from date palms.[28–30] Another major cause of brain injuries in Oman are the 'diseases of affluence'—diabetes, hypercholesteremia, and obesity—that lead to strokes.[31, 32] For victims of these, the protocol has limited itself to reducing mortality.

With a 'low child, low adult' mortality stratum, Oman's population has an extremely large youth base, with 65% below 25.[33] Recklessness, novelty seeking and proneness to accidents peak in adolescence, the underdeveloped frontal cortex in young people being cited as one of the many reasons for this.[34] An unusual trend in Oman is reckless driving in young women. Whereas studies from elsewhere place road traffic accidents in the 'male turf', for reasons not yet apparent, females in Oman have managed to blur such demarcation.[35]

With the advent of effective emergency care, faster transportation and acute medical management, the mortality rate among the brain-injured has decreased in Oman. However, merely saving lives and leaving them at that may result in a most distressing quality of life for the survivors who are even incapable of self-care.

Prevention being infinitely superior to rehabilitation, the youth of Oman should be intensely targeted from their early teens, and the dangers of reckless driving should become an important part of the campaign to reduce injuries and discourage lifestyle conducive to the 'diseases of affluence.' In 1998, 23 people per 1,000 vehicles were either maimed or killed in traffic accidents.[3] Psychosocial studies should be carried out to delineate behavioural factors leading to injuries and tease out culture-specific factors responsible for accidents.[36] As youth often occurs with a sense of invincibility, those more sober forces of society must assist in the prevention of the catastrophic. Traffic surveillance should be stepped up, backed by stiff and inescapable fines. Programmes should be evolved to wean away youngsters from the thrill of fast cars to sports or other healthier forms of catharsis relevant for youth.

In spite of best efforts at prevention, the numbers of cases of brain damage will keep rising at least in the short and medium term. During just one year from 1999 to 2000, debilitating injuries in Oman increased by 9%.[3] As we have discussed, emerging evidence gives hope that these unfortunate people can indeed be rehabilitated to regain self-sufficiency. This needs now to be a priority for healthcare planners in Oman who are urged to review and revamp the policies and facilities for rehabilitating the victims of physical, sensory, cognitive, developmental or emotional disabilities that have occurred due to brain injuries. We must move not one but two steps ahead of our current practices. We must encourage the primary prevention of the injuries, and then bring to bear the full weight of our medical system to address the issues described above.

We can and should see each individual as having a weakened neurochemical system that can be strengthened, as having rescue mechanisms that can be coerced

to accelerate, and once introduced, as having new tissue available which must be trained towards maximal function. These processes require medical diligence. It is time the healthcare system of Oman rose to the occasion and gave due importance to the rehabilitation of those with brain injury.

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