The Role of Fear in the Agonistic Complex (Part III)

by Johan M.G. van der Dennen

The Physiology of Fear and Anger (and Sex)

Cannon (1920 et seq.) found that when an animal was confronted with a situation which evoked pain, rage or fear, it responded with a set of physiological reactions which prepared it to meet the threat with ‘fight’ or ‘flight’. These reactions, said Cannon, were mobilized by the secretion of adrenalin: when the cortex of the brain perceived the threat, it sent a stimulus down the sympathetic branch of the autonomic nervous system to the adrenal glands and they secreted the hormone. Cannon graphically described the results as follows:

“Respiration deepens; the heart beats more rapidly; the arterial pressure rises; the blood is shifted away from the stomach and intestines to the heart and central nervous system and the muscles; the processes in the alimentary canal cease; sugar is freed from the reserves in the liver; the spleen contracts and discharges its content of concentrated corpuscles, and adrenalin is secreted from the adrenal medulla. The key to these marvelous transformations in the body is found in relating them to the natural accompaniments of fear and rage – running away in order to escape from danger, and attacking in order to be dominant. Whichever the action, a life-or-death struggle may ensue. The emotional responses just listed may reasonably be regarded as preparatory for struggle. They are adjustments which, so far as possible, put the organism in readiness for meeting the demands which will be made upon it. The secreted adrenalin cooperates with sympathetic nerve impulses in calling forth stored glycogen from the liver, thus flooding the blood with sugar for the use of laboring muscles; it helps in distributing the blood in abundance to the heart, the brain, and the limbs (i.e. to the parts essential for intense physical effort) while taking it away from the inhibited organs in the abdomen; it quickly abolishes the effects of muscular fatigue so that the organism which can muster adrenalin in the blood can restore to its tired muscles the same readiness to act which they had when fresh; and it renders the blood more rapidly coagulable.

The increased respiration, the redistributed blood running at high pressure, and the more numerous red corpuscles set free from the spleen provide for essential oxygen and for riddance of acid waste, and make a setting for instantaneous and supreme action. In short, all these changes are directly serviceable in rendering the organism more effective in the violent display of energy which fear or rage may involve”.

Cannon recognized that among all these physiological changes there were a few which could not be ascribed directly to the action of adrenalin. He therefore postulated that the hormone was supplemented by two additional substances from the sympathetic nerves. An active agent, distinguishable from adrenalin, was eventually identified in 1948. It proved to be a second hormone secreted by the adrenal medulla. Called noradrenalin, it differs markedly in its physiological effects. (Note: these substances are also called epinephrine and norepinephrine).

In recent years adrenalin and noradrenalin have been assigned central roles in attempts to account for physiological changes in fear and anger. Goldenberg et al. (1948) found the following reactions to adrenalin by normal subjects:
• Striking increase in cardiac output
• Significant rise in systolic blood pressure
• Insignificant rise in diastolic blood pressure
• Slight rise in mean arterial pressure
• Sharp drop in peripheral blood vessel resistance
• Moderate rise in pulse rate

The response of subjects to noradrenalin was somewhat different:

• No change or moderate increase in cardiac output
• Significant rise in systolic blood pressure
• Significant rise in diastolic blood pressure
• Significant rise in mean arterial pressure
• Striking increase in peripheral resistance
• Significant increase in pulse rate

Examination of these lists reveals two main differences. Adrenalin elevates cardiac output so that more blood is delivered; and it acts as a vasodilator, allowing the blood to reach skeletal muscles and decreasing peripheral resistance. Noradrenalin has little or no effect on cardiac output; and it acts as a vasoconstrictor, cutting off blood to the peripheral blood vessels that supply muscles. Thus adrenalin is an emergency stimulant with widespread and differentiated action, while noradrenalin is useful in the everyday vegetative process of blood pressure homeostasis (von Euler, 1955; Funkenstein, 1955; Buss, 1961).

In the face of danger the organism must prepare itself for sustained, vehement or violent activity: to run long enough to outdistance the pursuer or to attack with sufficient force to overcome the source of the danger: the ‘fight or flight’ pattern (Cannon, 1920 et seq.; Buss, 1961).

A person may experience three major emotions in response to a threatening situation: anger directed outward (the counterpart of rage), anger directed toward himself (depression) and anxiety, or fear. In studies of physiological changes accompanying various emotional states, Wolff and his coworkers (e.g. Wolff, Wolf & Hare, 1949) noticed that anger produced effects quite different from those of depression or fear. For example, when a subject was angry, the stomach lining became red and there was an increase in its rhythmic contractions and in the secretion of hydrochloric acid. When the same subject was depressed or frightened, the stomach lining was pale in color and there was a decrease in peristaltic movements and in the hydrochloric acid secretion. The experiments of Wolff, the evidence that the adrenal medulla secreted two substances rather than one, and certain clinical observations led a group at the Harvard Medical School (Funkenstein, King & Drolette, 1954; Funkenstein, 1955; 1956) to investigate whether adrenalin and noradrenalin might be specific indicators which distinguished one emotion from another. Their results suggested that anger directed outward was associated with secretion of noradrenalin, while depression and anxiety were associated with secretion of adrenalin.

In a classic experiment on anger and fear, Ax (1953; 1960) found seven of the fourteen frequency measures used to show significant differences between anger and fear. Anger was higher than fear in number of diastolic blood pressure rises, heart rate falls, galvanic skin responses and muscle tension increases; fear was higher than anger in skin conductance increases, muscle tension peaks, and respiration rate increases. These physiological
differences between anger and fear refute the notion that the ‘flight or fight reflex’ stems from a single pattern of physiological changes.

Ax attempted to relate his findings to the actions of adrenalin and noradrenalin. He suggested that the anger pattern resembles the changes that occur when both adrenalin and noradrenalin are injected, and the fear pattern resembles the changes that occur when adrenalin is injected (Buss, 1961). This indicated that the physiology was specific for the emotion rather than for the person (Funkenstein, 1955).

For the physiological differentiation of emotional states, notably fear and anger, see also: Landis & Hunt, 1939; Wolf & Wolff, 1942; Arnold, 1945; 1960; Mahl, 1949; Almy, 1951; Grace, Wolf & Wolff, 1951; Schneider & Zangari, 1951; Wolff, 1953; Davis, Buchwald & Frankman, 1955; Engel, Reichman & Segal, 1956; Elmadjian, Hope & Lamson, 1957; Funkenstein, King & Drolette, 1957; Schachter, 1957; Regan & Reilly, 1958; Gellhorn, 1960; Gellhorn & Loofbourrow, 1963.

Together with the studies of Funkenstein, King & Drolette (1954), Funkenstein (1955), Cohen, Silverman & Zuidema (1956), Silverman, Cohen & Zuidema (1957), Cohen & Silverman (1959), Schachter & Singer (1962), Schachter & Wheeler (1962), Schachter & Latane (1964), Schildkraut & Kety (1967), the results indicate a close link between anger, aggressive reactions, and noradrenalin level, while adrenalin has been associated with anxiety in fear-provoking and unpredictable situations (See Breggin, 1964, for a review on anxiety).

Cohen & Silverman (1959) attempted to tie in developmental considerations in their explanation of the results. They suggested that the relationship of specific affective constellations and catecholamine levels might have its basis in the association of certain infantile emotional responses and their physiological concomitants.

An infant’s reaction to unpleasant inner sensations associated with deprivation, frustration or pain can be perhaps characterized as a massive dedifferentiated discharge phenomenon. Neonates have high levels of noradrenalin and low levels of adrenalin. The high noradrenalin levels may in part be the result of this diffuse muscular activity. As infants develop their first attempts to cope with their increased tension this may be defined as primitive aggression. Hence, it is postulated that the affect of rage or anger or the carrying out of aggressive behavior might re-excite these circuits and lead to the release of noradrenalin even in the absence of the diffuse muscular activity which was the original stimulus calling forth the noradrenalin release. As muscular coordination increases, this ability might be used in the service of goal directed activity. Thus, the hormonal reaction may be similar to that of the infant in a primitive rage state although the affect perceived is far less intense, and the muscular activity less diffuse (Buss, 1961).

The entire series of experiments listed above yield data which can be understood in the frame of reference of psychoanalytical observations. According to this theory, anger directed outward is characteristic of an earlier stage of childhood than is anger directed toward the self or anxiety (conflicts over hostility). The latter two emotions are the result of the acculturation of the child. If the physiological development parallels its psychological development, then we should expect to find that the ratio of noradrenalin to adrenalin (NA/A ratio) is higher in infants than in older children. Hokfelt (1951), Shepherd & West (1951), and West, Shepherd & Hunter (1951) established that this is indeed the case: at an early age the adrenal medulla has more noradrenalin, but later adrenalin becomes dominant.
Outward aggression – a circular, self-perpetuating phenomenon – and concomitantly high NA/A ratio may continue in adolescent and adult life as a function of differential child rearing practices, i.e. physical punishment and subsequent outward expression of aggression.

Fine & Sweeney (1967) found a significant relationship between biographical variables used as indices of socio-economic status and the NA/A ratio. Their results support the original work of Funkenstein, King & Drolette (1957), who presented evidence that direction of expression of aggression was related to parental strictness and dominance, and to social class.

Von Euler (1955; 1959) compared the adrenal secretions found in a number of different animal species. Aggressive animals had a relatively high amount of noradrenalin, while in animals such as the rabbit, which depend for survival primarily on flight, adrenalin predominated. Domestic animals, and wild animals that live very social lives also have high a ratio of adrenalin to noradrenalin.

Kinsey et al. (1953) has pointed out that nearly all of the physiological elements of the sexual response are found in other situations, particularly in other emotional responses and most particularly in anger. They present the following table:

<table>
<thead>
<tr>
<th>Physiologic element</th>
<th>Sex</th>
<th>Anger</th>
<th>Fear</th>
</tr>
</thead>
<tbody>
<tr>
<td>increase in pulse rate</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>increase in blood pressure</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>vasodilation</td>
<td>+</td>
<td>may</td>
<td>_</td>
</tr>
<tr>
<td>increased peripheral circulation</td>
<td>+</td>
<td>may</td>
<td>_</td>
</tr>
<tr>
<td>tumescence</td>
<td>+</td>
<td>rare</td>
<td>_</td>
</tr>
<tr>
<td>reduced rate of bleeding</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>hyperventilation</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>breathing irregularity</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>diminished sensory perception</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>increase in genital secretions</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>increase in salivary secretion</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>increase in lacrimal secretion</td>
<td>+</td>
<td>?</td>
<td>?</td>
</tr>
<tr>
<td>increase in perspiration</td>
<td>+</td>
<td>?</td>
<td>+</td>
</tr>
<tr>
<td>adrenaline secretion</td>
<td>?</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>increase in muscular tensions</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>increase in muscular capacity</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>involuntary muscular activity</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>rhythmic muscular movements</td>
<td>+</td>
<td>?</td>
<td>-</td>
</tr>
<tr>
<td>gastro-intestinal activity inhibited</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>hair raised (piloerection)</td>
<td>may</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>eye pupil dilated</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>involuntary vocalization</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>ejaculation</td>
<td>+</td>
<td>rare</td>
<td>rare</td>
</tr>
</tbody>
</table>

One might hypothesize that if certain of the physiologic elements were prevented from developing in a sexual response, or taken away from a sexual response, the individual might
be left in a state of anger or fear, or in some other emotional state. The fact that frustrated sexual responses so readily turn into anger and rage might thus be explained. On the other hand, it not infrequently happens, both in the lower mammals and in man, that anger, fighting and quarrels suddenly turn into sexual responses. The closest parallel to the picture of sexual response is found in the known physiology of anger. The table shows fourteen items which usually appear in both situations. There are only four respects in which the physiology of anger does not fit the known physiology of sex: tumescence, genital secretions, rhythmic muscular movement, and orgasm. While penile erections may sometimes appear in anger, particularly among the infrahuman mammals and among pre-adolescent human males, they are not usual (Kinsey et al., 1953).

**Variables Affecting the Physiology of Anger and Fear**

When adrenalin or noradrenalin is injected into the body, there is a lapse of time before the substance can be circulated throughout the body to target sites. The same is true when either substance is secreted by the adrenal medulla into the blood stream. Schachter (1957) reported that in many subjects heart rate and galvanic skin response attained their peak changes within three seconds of the onset of the anger stimulus. While the action of chemicals circulating in the blood would require a longer interval than 3 seconds, neural mediation could easily occur in less time. Therefore, it would seem that the first changes in fear and anger are mediated by sympathetic nervous system activity. With the passage of time the physiological picture becomes complicated by the action of homeostatic mechanisms. Initial vasoconstriction that leads to higher blood pressure will activate reflex vasodilator mechanisms like the carotid sinus reflex, which insures that blood flow to the brain does not become excessive. Thus, even if the physiological response to anger were initially uncomplicated, it would appear complicated as soon as homeostatic mechanisms began to operate.

Other complicating variables are the intensity of anger and individual differences. First, there is a wide variability in resting level of blood pressure, pulse rate, gastric activity, etc.

A second kind of variability involves differences in extent of change. It is well known that individuals differ in physiological reactivity, some having somewhat more labile visceral reactions than others.

A third kind of variability involves individual differences not in resting level or lability but in the pattern of physiological response elicited by a given stimulus (Buss, 1961).

**Stress**

Selye (1956), perhaps the foremost student of the physiology of stress, states that “in its medical sense, stress is essentially the rate of wear and tear in the body. Anyone who feels that whatever he is doing – or is being done to him – is strenuous and wearing, knows vaguely what we mean by stress”.

Selye has determined that man reacts to stress-inducing agents or stressors through what he terms the ‘General Adaptation Syndrome’. This syndrome is composed of three identifiable stages. The first is the alarm reaction period in which the organism is alerted to the existence of stress. Since no organism can be maintained permanently in a state of alarm, a second state,
that of resistance, emerges in which adjustment and tolerance to the stress takes place. If stress continues it may overburden the capability of the organism to adjust, and the third stage, that of exhaustion, is reached in which the organism can no longer tolerate the stress.

Chronic stress, Dobzhansky (1962) has emphasized, “overtaxes the body’s stamina and may lead to nervous and physical breakdown and finally to death”.

There has persisted in man a cluster of hormonal and metabolic characteristics which have developed as a response to threatening, perhaps even crisis, situations in nature. These human physiological responses to stress were first described in minute detail in the now-classic work by Cannon (1929), which deserves to be quoted at length:

“The adrenin (adrenalin) secreted in times of stress has all the effects in the body that are produced by injected adrenin. It cooperates with sympathetic nerve impulses in calling forth stored carbohydrate from the liver, thus flooding the blood with sugar; it helps in distributing the blood to the heart, lungs, central nervous system and limbs, while taking it away from the inhibited organs of the abdomen; it quickly abolishes the effects of muscular fatigue; and it renders the blood more rapidly coagulable. These remarkable facts are, furthermore, associated with some of the most primitive experiences in the life of higher organisms, experiences common to all, both man and beast – the elemental experiences of pain and fear and rage that come suddenly in critical emergencies. What is the significance of these profound bodily alterations? What are the emergency functions of the sympathico-adrenal system?

The facts at once have significance if considered in relation to the struggle for existence. For ages past, this struggle has disciplined and relentlessly selected the most efficient. If fear always paralyzed it would result only in danger of destruction. But fear and aggressive feeling as anticipatory responses to critical situations, make ready for action and thereby they have had great survival values. And the remarkable system of internal adjustments which attend these emotions and which mobilize the forces of the body are such as to have had great survival values. Thus the bodily changes may reasonably be interpreted”.

Though his work has been amplified by others, notably Selye, the basic scientific validity of Cannon’s findings has withstood the test of scrutiny very well over the years.

For what purposes might natural processes have endowed the human organism with so complex a physiological adjustment process to external stresses? “It seems reasonable to conclude” states Hamburg (1961) “that the increased secretion of adrenal hormones under psychological stress facilitates energy mobilization involving increased availability of carbohydrate and fat for burning. This makes sense as it is viewed as serving a preparatory function for the oxidative process required in muscular exertion”.

Thus, early man, confronted as he was by the multitude of threats to his personal safety from his natural environment, was prepared for action by a set of physiological anticipatory preparations. Many writers have described this as the ‘fight or flight mechanism’. It should be pointed out that the anticipated end result of this process was some type of physical activity which was predetermined by the massive infusion of blood into the muscular structure. The threatened individual either fled his challenger or he remained and fought. In either case, muscular exertion, as the physiological outcome of the internal anticipatory preparations, was a natural and cathartic result (Wiegele, 1973).
It has been pointed out by many authors that in modern times this biological equipment has become obsolete and perhaps even maladaptive (Buss, 1961; Hamburg, 1961; Dubos, 1965; Wiegele, 1973; among others). The situation of a person physiologically mobilized with no prospect of physical action has been well summarized by Dubos (1965):

“Since the circumstances which stimulate the endocrine system to mobilize the bodily resources rarely demand the actual expenditure of physical energy in the modern world, the usual result is that physiological preparation for action is not followed by action. The failure to act after the body has achieved a state of physiological and metabolic readiness constitutes a disturbing biological experience which is extremely common in civilized life, often frustrating, and likely to be deleterious”.

Physiological Arousal and Cognitive Content

Marañon (1924) first described the subjective perception in some persons who were administered adrenalin (epinephrine) as an ‘as if’ or ‘cold emotion’: they lacked the psychological component of the emotion. The effects of adrenalin could be potentiated by thyroid hormone, a finding consistent with many subsequent studies (Barchas et al., 1972).

Cantril & Hunt (1932) showed, in their experiments with adrenalin administration, that the situation must contain certain logical cognitive relationships for the experience to contain an element of genuine emotion.

Of tremendous theoretical and experimental interest have been the studies of Schachter & Singer (1962) and Schachter & Wheeler (1962). Their results justified the conclusion that the emotional tone is not directly related to the degree of activation, but rather to the cognitive accompaniments of the activation, and that without any prior information, the subjects will attempt to fix a cognition to explain the physiological activation.

Their general conclusion was that emotional state is a function of a state of physiological arousal and an appropriate cognition.

Neural, Neuroendocrine and Neurochemical Systems Involved

Evidence from Neuropathology

Neuropathological changes in the central nervous system (CNS) – so-called substrate changes, particularly lesions and brain neoplasms – may result in disruptions of behavioral, motivational and/or emotional systems, and chronic personality changes. Many of these syndromes present a clinical picture in which fear and aggression – ranging from enhanced irritability and deleterious mood changes, dysphoric states, hostility, to all-out violent attack – are conspicuous symptoms.

Van der Dennen (1983) presented a review of the literature on pathological aggression associated with organic brain disorders, i.e. disorders on the basis of discernible
neuropathology of the CNS, such as temporal lobe epilepsy, brain neoplasms, cerebral trauma, minimal brain dysfunction, rabies, encephalitis, and abnormal electroencephalogram.

Furthermore, those neuroendocrinological and metabolic disorders which affect the CNS and result in enhanced aggression were reviewed, such as the premenstrual syndrome, hypoglycemia, allergic tension-fatigue syndrome, Lesch-Nyhan syndrome, phenylketonuria, and others.

Among the major conclusions are that (1) a variety of neural substrate changes, preferentially involving the limbic system, may result in pathological aggression, most probably by damaging the fight/flight neural circuitry; (2) lesions of the CNS may give rise to at least two phenomenologically distinct kinds of pathological aggression:

(a) paroxysmal rage outbursts of an impulsive, relatively uncontrollable nature, related to an ‘offensive aggression’ circuitry in the limbic system; and (b) self-defensive striking-out and assaultiveness due to delusional beliefs, hallucinations, and paranoid ideation, preceded and accompanied by overwhelming emotions of fear and terror. The eventual violence used is essentially self-defensive and panicky – the preemptive strike of a ‘cornered cat’.

This conclusion confirms the distinction in offensive and defensive systems as reported in many animal studies (Cf. Adams, 1979; 1980; Blanchard et al., 1984). It also points to the conspicuous role of fear in human interpersonal violence.

Evidence from Neuroendocrinology

Fearful experiences, such as a severe defeat in an agonistic encounter, may have profound and relatively long-lasting influences on the endocrine system. Fear reduces, for instance, the testosterone level in male rats (Bermond, 1977), and with it probably the tendency to put up resistance when provoked. Long intervals between agonistic encounters allow this and other hormone levels to return to baseline level, but when the intervals between the fearful experiences are too short, the effects accumulate, thus rendering the individual inclined to flee instead of to put up resistance when challenged again. A considerable amount of evidence is available to support this idea.

For instance, Eleftheriou & Church (1968) demonstrated that in mice the blood plasma levels of LH (luteinizing hormone), LH-RF (LH releasing factor) and FSH (follicle stimulating hormone) decreased after aggressive encounters. The hormonal changes in losers appeared to be cumulative in contrast to the hormonal changes in winner-mice.

Rose, Gordon & Bernstein (1972) demonstrated similar hormonal effects in rhesus monkeys. Schuurman (1977) found that aggressive encounters brought about stronger physiological changes (in blood sugar level, inter alia) in loser rats than in winner rats.

In more anthropomorphic terms: A conflict or encounter which elicits emergency reactions and raises anxiety in the subordinate may well represent an ordinary – perhaps even exciting – matter of routine to the dominant. All this means that apart from opposite conditioning effects of agonistic encounters on winners and losers on the neural level, a stable behavioral divergence between the social roles is still further enhanced and consolidated by different hormonal adaptations.
Similar effects have also been demonstrated in humans. Kreuz, Rose & Jennings (1972) conducted physiological studies on trainees in a military camp and found that in periods of high tension, stress and extremely hard work, their testosterone level decreased considerably and recovered only gradually after relaxation opportunity.

Different physiological changes have also been observed after shock-induced fighting or escape behavior in rodents (Williams & Eichelman, 1971; Conner, Vernikos-Danellis & Levine, 1971). Conner et al. suggest that these different physiological changes (e.g. decreased blood pressure and ACTH secretion) represent an adaptive consequence of fighting, in that they lessen the internal response to the stressor. Fighting behavior could be indirectly affected by testosterone and similar steroids through an attentional change, thereby making attack more persistent and hence prolonged. Furthermore, androgens are anabolic, promoting muscular development, which would enable an animal to be more successful in a fight. Bronson & Desjardins (1971) found that defeat depresses gonadal activity.

Hormones of the pituitary-adrenocortical axis may also be involved. ACTH appears to have an inverse effect on the readiness to attack in rodents, low levels causing increased aggressiveness (Candland & Leshner, 1974; Leshner et al., 1973). Various procedures, including prolonged social isolation, lower basal adreno-pituitary activity (Welch & Welch, 1969), and thus influence aggressiveness, either continuously or as a step function of threshold effects (See Archer, 1976).

It has been suggested, furthermore, that pituitary-adrenal manipulation indirectly effects the readiness to attack by influencing the level of fear (Brain, 1972; Sware & Leshner, 1973; Candland & Leshner, 1974; Leshner, 1975), or facilitating flight by releasing adrenalin from the adrenal medulla (de Wied, 1966; Laborit et al., 1975). Thus, ACTH would decrease aggressiveness by increasing the readiness to show fear behavior in the presence of novel, potentially aggressive conspecifics, and thereby predispose the animal to be more submissive (Leshner, 1975; Archer, 1976). Following competition, defeated animals show increased levels of adrenocortical activity, while victorious animals seem unaffected (Archer, 1970; Bronson & Eleftheriou, 1964; 1965a). In fact, the mere threat of defeat is sufficient to elicit increases in adrenocortical activity in previously defeated mice (Bronson & Eleftheriou, 1965b). Subordinate rodents, living under the constant threat of defeat or subjected to repeated defeats, have higher levels of pituitary-adrenocortical activity than dominant rodents (Davis & Christian, 1957; Louch & Higginbotham, 1967; Southwick & Bland, 1959).

Fighting leads to decreased serum levels of both LH and FSH, and the magnitude of this depression of gonadotrophin levels is greater in defeated than in victorious mice (Bronson, Stetson & Stiff, 1973). The temporal characteristics of the gonadotrophin responses to competition also are different for defeated and victorious mice: FSH levels remain depressed longer following fighting in defeated mice than in victorious mice. However, LH levels remain depressed for long post-experience time periods, and the outcome of the fight does not influence the rate of return of LH levels toward normal levels (Bronson, 1973). Agonistic experiences thus produce dramatic changes in the state of the CNS circuits which control endocrine function (Leshner, 1975). Defeat has a significant effect on protein synthesis in the brain (Eleftheriou, 1971) and alters the brain neurotransmitter systems. Daily fighting experiences lead to increases in central catecholamine and serotonin levels (Welch & Welch, 1969; Modigh, 1973).
Non-aggressiveness (predicting submissiveness) can be experimentally produced by sustained elevations in ACTH levels (Leshner, 1981). However, the available evidence suggests that in the case of submission, the critical pituitary-adrenal hormone is corticosterone, not ACTH, as the studies on aggression would predict. The experience of defeat leads to marked increases in corticosterone levels in mice (Bronson & Eleftheriou, 1965; Leshner, 1980). Leshner (1981) hypothesized that the greater the corticosterone response to an initial defeat experience, the greater or more intense is the stimulus complex surrounding that defeat experience, and, therefore, the more submissive the animal will be in future encounters. Furthermore, there is some evidence of endogenous opioids (endorphins) to be involved in submissiveness (Miczek, Thompson & Schuster, 1982).

When male rhesus monkeys are introduced as ‘strangers’ into established captive groups they are attacked and often wounded. Such males experience a pronounced decline in circulating testosterone, whereas hormone levels may increase in dominant aggressive males (Rose, Gordon & Bernstein, 1972; Rose, Bernstein & Gordon, 1974). Under laboratory conditions, Perachio (1978) found that plasma testosterone levels rose dramatically in both dominant and subordinate males that exhibited fighting behavior. Keverne, Miller & Martinez-Arias (1978) suggested that changes in peripheral levels of testosterone which follow aggressive interactions reflect alterations in neurotransmitter activity in the brain (Dixson, 1980).

Leshner (1975) has argued that, if an animal’s initial experience is defeat, its hormonal characteristics will change in the direction of nonaggressive animals, and, therefore, its behavior should become less and less aggressive and more and more submissive. According to this view, initial experiences in agonistic situations feed back through the endocrine system to alter the animal’s perception of the agonistic stimuli and, therefore, its behavior.

It is also possible that these initial responses to defeat produce long-term modifications in the animal’s hormonal state so that future agonistic reactions will be modified as a function of prior experiences. Early exposure to defeat predisposes an animal to be nonaggressive in adulthood (Kahn, 1951), and prior experiences of defeat in adulthood condition an animal to be more subordinate the next time it encounters an agonistic situation (Scott & Marston, 1953).

It is possible that the hormonal responses to defeat are sufficiently long-lasting that a previously-defeated animal enters future agonistic situations in a different baseline hormonal state from inexperienced animals. Thus, the previously-defeated animal would perceive the agonistic stimuli as more fear-provoking, and its behavior would be more submissive and less aggressive. Thus it is likely that the endocrine responses to competition provide at least three mechanisms for adapting to the stresses of competition, particularly defeat: a physical mechanism of adaptation to stress, and a behavioral change and a stimulus quality change which serve to reduce the amount of stress to which the animal is exposed (Leshner, 1975).

In humans, anxiety is associated with higher levels of adrenalin, while outward-directed anger is associated with higher levels of noradrenalin (Funkenstein, 1955; Gray, 1972; Vide supra). It should be emphasized in this context, however, that previous experience can override such hormonal influences on aggressiveness even in rodents. In gonadectomized talapoin monkeys, previous social experience and acquired learning of hierarchic rules are more important than sex hormones in aggressiveness and dominance pattern formation (Dixson & Herbert, 1977).
Apart from the neuroendocrine feedbacks as depicted above, differential learning processes on the neural level are involved as well. Animals in a βeta-role and animals in an alpha-role experience aggressive encounters in a different way, and therefore different neural information is stored and processed. Besides, as e.g. Buchholtz (1978) points out, there is a great functional and physiological difference between short-term memory storage and long-term memory storage. And there are limits to the amount of experiences of a certain type pro time unit that an individual can successfully ‘digest’ (Van der Molen & Van der Dennen, 1981) and transform into enhanced long-term skills. This phenomenon has been established in a wide range of species from insects to man. The neural and hormonal feedback systems may be considered to function more or less synergistically.

The limbic system (Papez, 1937; MacLean, 1949 et seq.), usually considered to control affectivity, plays an essential role in the establishment of long-term memory (Milner, Gorkin & Teuber, 1968), without which affectivity would be impossible. This is because long-term memory – related to protein synthesis (Hyden & Lange, 1968) – is necessary if a given previously experienced situation is to be identified as agreeable or disagreeable, so that the appropriate ‘affect’ can be triggered by it (Laborit, 1978). In the case of a ‘novel’ situation that, because of an information deficit is not immediately classifiable, memory again plays a crucial role in sorting the experience as potential threat or agreeable experience.

Long-term memory thus makes possible temporal and spatial association, within the synaptic circuits, of memory traces identified with experience-related, information-bearing signals, which in turn facilitate the development of conditioned reflexes, either the Pavlovian affective or vegetative type, or the Skinnerian neuro-motor variety (Laborit, 1978).

In a similar vein, Van der Dennen (1980) has argued that relatively long-term attitude-like human sentiment-structures like hatred, vengefulness, rancorous resentment, vindictiveness, hostility, etc. would be quite unthinkable without long-term memory, enabling the individual to recall and ruminate on previous humiliating experiences (Cf. also Zillmann, 1979). Man ‘incorporates’ his enemies so to speak.

According to Laborit (1978), defensive aggression might seem to bear some similarity to fear-aggression, but the latter differs in that it involves learned punishment expectation. Fear presupposes a knowledge of the existence of disagreeable stimuli and the knowledge that situations previously identified as such require evasive action in the form of flight or confrontation. There will always be some situations, however, that are so unusual that they will not be covered by previous experience and will therefore not be identifiable as either painful, neutral or gratifying. This results in action inhibition accompanied by anxiety feelings, rather than fear. Only if aggressive behavior has previously been used successfully in analogous situations will it perhaps be used again, preventively.

Antagonistic systems called the System of Action Inhibition (SAI) and the System of Action Activation (SAA) have been described by Laborit (1974; 1975; 1978). In both animals and man, disturbance of the internal biological equilibrium leads to more or less instinctive behavior patterns designed to satisfy endogenous needs arising from instinctive cravings triggered by hypothalamic stimulation.

If the action taken is rewarded and the need met, then a memory trace will subsist, reinforcing and facilitating repetition of the behavioral strategy employed. This system is catecholaminergic. If, on the other hand, the action taken is not rewarded, or is punished, this
will trigger a behavioral pattern of flight or, if this too is ineffective, of confrontation or defensive aggression. This behavior pattern also activates various cerebral levels, but it does so through the periventricular system (PVS) which is cholinergic.

However, if the behavior is either punished or left unrewarded, or if both escape and confrontation prove ineffective, then an inhibition behavior pattern, suppressing an acquired behavior pattern, will intervene. The system of action inhibition (SAI) activates the median septal area, the dorsal hippocampus, the caudate nucleus, the lateral amygdala and the ventro-median hypothalamus; it is cholinergic and possibly serotonergic as well. Associated with the activation of these various areas and circuits of the CNS are certain endocrine actions, among which those involved in the alarm system (Selye, 1936), particularly the pituitary-adrenocortical axis, controlled by Corticotrophin Releasing Factor (CRF).

The system identified by de Molina & Hunsperger (1962), the PVS, controls flight and confrontation behavior and is cholinergic. When activated, it triggers the release of the CRF and the secretion of ACTH. But if the behavioral strategy employed is effective and leads to gratification, then the secretion of ACTH and subsequent release of glucocorticoids will both be arrested. The ACTH released immediately following aggression stimulates the system of action activation (SAA) (Bohus & Lissak, 1968). When ACTH is injected into a normal animal a considerable quantity of adrenalin is released from the adrenal medulla (Laborit et al., 1975). ACTH facilitates flight and confrontation behavior and also defensive aggression (de Wied, 1966), because adrenalin is a vasodilator in the organs crucial for the individual’s motor autonomy in the environment. However, behavior that is unrewarded will be inhibited by the cholinergic SAI and secretion of the various endocrine factors will continue unchecked. In addition, we now know that the glucocorticoids themselves stimulate repression of inhibitory behavior. There results a kind of vicious circle that can be broken only by either effective action or disappearance of the punishment.

Injection of hydrocortisone into adrenalectomized animals causes the release of considerable amounts of noradrenalin into the plasma through the nerve ends of the peripheral sympathetic system (Laborit et al., 1975). These observations have led to the conclusion that stimulation of the SAI is responsible for this. The PVS stimulates medulloadrenal secretion of adrenalin with a vasodilator effect on the organs of motor activity in the environment. Noradrenalin, which is vasoconstrictive relative to all smooth vascular fibers, appears on the other hand to be the neuro-hormone of crisis reaction, when mobility of the individual in the environment is crucial.

When gratification is not obtained and when flight and confrontation both prove ineffective against aggression, a behavior pattern of motor inhibition results. Defeat is preferable to combat that might end in death. But this pattern also creates a vicious circle with, on the vegetative level, considerable increase in noradrenalin circulation and, on the endocrine level, the release of glucocorticoids that stimulate the system of action inhibition. This leads to a state of tension that can be resolved only by gratification and that sometimes will lead either to explosions of aggression or to a state of depression. This state of tension is also responsible for the condition generally known as psychosomatic diseases (like neurogenic hypertension and gastric ulcers). Laborit would prefer the term ‘diseases of behavioral inhibition’.

Several researchers have pointed to the hypothetical functional similarity of ‘fear’ and ‘rage’ (flight and attack), suggesting that similar immediate causal factors can evoke either emotion/behavior pattern, and that both flight and attack might serve the similar function of
restoring a dynamic equilibrium (Hebb, 1946; Scott & Fredericson, 1951; Berkowitz, 1962; Hinde, 1969; 1970). Galef (1970) and Archer (1976) suggested that attack and avoidance behavior might be two varieties of a single type of response to novelty, though not necessarily based on a single neurophysiological system. At least in some components the motor systems of attack and flight are antagonistic.

**Fear in Human Aggression and Violence**

That fear may play a prominent part in several forms and types of human aggression and violence, has not gone unnoticed by students of human behavior, though the analysis seldom goes beyond frequent references to the cornered cat that makes vehement jumps – the fear-aggression/violence relationship has hardly been systematically investigated. Yet, in the theoretical literature sufficient angles can be found. For instance, Carp (1967), following Bovet (1928), defined aggressiveness explicitly as “la peur en avant”.

In Vestdijk’s (1979) analysis of fear, he concludes that no aggression is possible in humans without underlying fear.

To Lloyd George is attributed the dictum that “It is the fears of nations that makes conflict”. Similarly, J.G. Miller (1951) stated: “Ignorance of the desires, aims and characteristics of other peoples leads to fear and is consequently one of the primary causes for aggression”. Criticizing such a notion, ‘t Hart (1957) remarked that ignorance of other peoples does not necessarily lead to fear.

“People’s fear forms the main source of aggressiveness. It is fear for the own prestige, fear to be overrun, fear to give anything whatever to an opponent, let alone whatever he may claim as his due right” (Rombouts, 1938).

“Fear and a sense of inferiority are great stimulants of aggressiveness in nations as in individuals. Projection of my own repressed aggression on to the other man produces the conviction that he means to attack. Therefore I must be prepared to defend myself, but only, of course, because I know I am going to be attacked. And so with nations” (Browne, 1938).

“Abundant clinical material has taught us that panic is a potent trigger for violence, and extreme fear of an adversary is no less likely to provoke an aggressive act against him than is hatred of him. Indeed fear is often the real basis for the hatred and is therefore the more fundamental motivation” (Marmor, 1964).

“Hostility grows out of fear... Fear of being blocked or harmed in some way is a major stimulus to violent action” (Stagner, 1967).

“One of the most powerful emotions leading to hatred and destruction is fear. It spreads like bush-fire and appeals to the most primitive instinct of self-preservation and to the archaic patterns of self-defense. The next step occurs when this emotion centers on an object of old resentment and suspicion, which then becomes doomed to destruction” (Bychowski, 1968).

“Konkretisierung der eher diffusen, unartikulierbaren Angst an einem dafür in der Tat geeigneten Realitätsausschnitt und die nachfolgende Agression als Antwort auf die Bedrohung, gehört zu geläufigen Psychopathologie der Sündenbockjagd. Das
Ineinandergreifen von diffuser Angst und Aggressionsbedürfnis einerseits, von gesamtgesellschaftlichen, auch ökonomisch vermittelten Bereitschaft zum Krieg andererseits ist deshalb so gefährlich, weil infantile Abwehrformen und objektive irrationalität einander eskalierend in die Hände spielen” (Horn, 1969).

“Er is namelijk nog een andere factor die in de oorlogsbereidheid een belangrijke rol speelt, en wel de angst. Op het eerste gezicht schijnt dat vreemd. Wij zijn gewend haat, woede en agressiviteit als motivatie tot agressie te beschouwen en angst als motivatie tot vlucht. Tot op zekere hoogte is dit ook juist. Maar psychobiologisch onderzoek, zowel bij dieren als bij mensen, leert dat de verhoudingen reeds bij individuele conflicten gecompliqueerder zijn, en zeker ook bij groepsconflicten. Wanneer nl. bij een conflict tussen een sterker en zwakker dier (waarbij dit laatste wil vluchten) de vlucht onmogelijk wordt gemaakt (bv. wanneer beide dieren in een kooi of klein territorium worden samengebracht) valt het zwakker dier wel degelijk aan. Hetzelfde kennen wij bij mensengroepen die eerst trachten te ontsnappen uit een oomsingeling, doch wanneer dat niet lukt juist zeer gevaarlijk agressief kunnen worden, en dan z.g. ‘met de moed der wanhoop’, ‘met de rug tegen de muur’ vechten en doden” (Groen, 1974).

Fear may be either illusory or real, but in both cases the Thomas theorem applies: Whatever people define as real, will be real in its consequences.

The Rage of Impotence (Based on Mansfield [1991: 198-207])

In analyzing ‘violent men’ Toch and his colleagues found certain psychological and social characteristics common to all of them. They were all men who had ‘been flooded all [their] life with strong feelings of not being able to be’ what they should be, who felt ‘unsure’, weak and insignificant, ‘helpless’, ‘easily panicked’, ‘precarious’ and fearful (Toch, 1969: 138 et passim). While they were deficient in verbal and other social skills, they were distinguishable from others who had the same deficiencies because they feared being mistaken for a coward and victimized, were perpetually on guard against being denigrated and belittled, and were always on the alert for ‘another danger lurking around a nearby corner’. Their violence was, in fact, an expression of an inability to cope; it was blind rage, a frantic, ‘desperate, lashing-out’, in an effort to obliterate the situation, to destroy the other person. The satisfaction was always ‘the removal of the irritation’, so that life was now (it was to be hoped) ‘silent and peaceful again’. What is unwittingly being described is essentially the annihilating aggressiveness of the trapped and frightened animal.

For all animals (including the most timid and least predatory), there is one point at which they will fight. This is the point at which they find flight (the normal response to aggression) impossible, so that the ‘choice’ is between accepting destruction passively or fighting in an attempt to avoid death. In that situation the titmouse or the squirrel will turn into a whirlwind of viciousness, lashing out against the threatening predator with tooth and claw. Such aggression is cold and deadly: it seeks to annihilate the danger, to either remove it entirely or make escape possible. In such a situation even the mildest animal is capable of prodigies of violence.

The annihilating aggression of the endangered is broader, more complex, and potentially more easily misdirected than the focused, appetitive aggression connected with hunger for food, sex, or shelter. For one thing, it can involve a functional definition of ‘self’ that extends
beyond the actual biological organism to include the dependent offspring. Thus the female of most species (and the male in some) will fight to the death in the defense of endangered young, even in cases where parental flight is possible. The threatened death of the young is responded to ‘as if’ it were the death of the protecting adult.

Equally important is the fact that an animal can perceive aggression in a situation and respond with annihilating rage when in fact no actual threat to its existence is being posed. Defensive aggression, after all, rests on response, often genetically programmed, to certain signs, regardless of the other’s intent.

That humans share such a response with their animal brethren has long been known to military leaders. As early as 500 B.C. Sun Tzu, the great Chinese military theorist, made it explicit in his strategic principle of the ‘Death Ground’, that is, a position from which it is impossible to retreat. Throw the soldiers into a situation where there is no escape, Sun Tzu wrote, “and they will display the immortal courage of Chuan Chu and Ts’ao Kuei”.

Unfortunately, the supposedly greater intelligence of humans does not reduce the chances of misperceiving another’s intent; instead consciousness (carrying the capacity for projection and fantasy) intensifies the probability of misunderstanding. Indeed, once violence between human beings has come to seem normal or natural in a culture or subculture, the possibility is increased that another human may be perceived as dangerously threatening to one’s existence. Moreover, the human sense of ‘self’ may be broadened by socialization to include a particular self-image (as well as one’s tribe, clan, or nation). Thus simple biological survival can be less important than, and may even be sacrificed to, the need to protect the sense of one’s self (and group) as powerful, honorable, righteous, and in control of things and events.