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CHAPTER 2

Emotion and the autonomic nervous system: a prospectus for research on autonomic specificity

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Abstract: The question of whether there are different patterns of autonomic nervous system responses for different emotions is examined. Relevant conceptual issues concerning both the nature of emotion and the structure of the autonomic nervous system are discussed in the context of the development of research methods appropriate for studying this question.

Are different emotional states associated with distinct patterns of autonomic nervous system (ANS) activity? This is an old question that is currently enjoying a modest revival in psychology. In the 1950s autonomic specificity was a key item on the agenda of the newly emerging discipline of psychophysiology, which saw as its mission the scientific exploration of the mind–body relationship using the tools of electrophysiological measurement. But the field of psychophysiology had the misfortune of coming of age during a period in which psychology drifted away from its physiological roots, a period in which psychology was dominated by learning, behaviourism, personality theory and later by cognition. Psychophysiology in the period between 1960 and 1980 reflected these broader trends in psychology by focusing on such issues as autonomic markers of perceptual states (e.g. orienting, stimulus processing), the interplay between personality factors and ANS responsivity, operant conditioning of autonomic functions, and finally, electrophysiological markers of cognitive states. Research on autonomic specificity in emotion became increasingly rare.

Perhaps as a result of these historical trends in psychology, or perhaps because research on emotion and physiology is so difficult to do well, there

exists only a small body of studies on ANS specificity. Although almost all of these studies report some evidence for the existence of specificity, the prevailing *zeitgeist* has been that specificity has not been empirically established. At this point in time a review of the existing literature would not be very informative, for it would inevitably dissolve into a critique of methods. Instead, what I hope to accomplish in this chapter is to provide a new framework for thinking about ANS specificity, and to propose guidelines for carrying out research on this issue that will be cognizant of the recent methodological and theoretical advances that have been made both in psychophysiology and in research on emotion.

Emotion as organization

From the outset, the definition of emotion that underlies this chapter should be made explicit. For me the essential function of emotion is organization. The selection of emotion for preservation across time and species is based on the need for an efficient mechanism than can mobilize and organize disparate response systems to deal with environmental events that pose a threat to survival. In this view the prototypical context for human emotions is those situations in which a multi-system response must be organized quickly, where time is not available for the lengthy processes of deliberation, reformulation, planning and rehearsal; where a fine degree of co-ordination is required among systems as disparate as the muscles of the face and the organs of the viscera; and where adaptive behaviours that normally reside near the bottom of behavioural hierarchies must be instantaneously shifted to the top.

Specificity versus undifferentiated arousal

In this model of emotion as organization it is assumed that each component system is capable of a number of different responses, and that the emotion will guide the selection of responses from each system. Component systems differ in terms of the number of response possibilities. Thus, in the facial expressive system a selection must be made among a limited set of prototypic emotional expressions (which are but a subset of the enormous number of expressions the face is capable of assuming). A motor behaviour must also be selected from a similarly reduced set of responses consisting of fighting, fleeing, freezing, hiding, etc. All major theories of emotion would accept the proposition that activation of the ANS is one of the changes that occur during emotion. But theories differ as to how many different ANS patterns constitute the set of selection possibilities.

At one extreme are those who would argue that there are only two ANS patterns: 'off' and 'on'. The 'on' ANS pattern, according to this view, consists

of a high-level, global, diffuse ANS activation, mediated primarily by the sympathetic branch of the ANS. The manifestations of this pattern — rapid and forceful contractions of the heart, rapid and deep breathing, increased systolic blood pressure, sweating, dry mouth, redirection of blood flow to large skeletal muscles, peripheral vasoconstriction, release of large amounts of epinephrine and norepinephrine from the adrenal medulla, and the resultant release of glucose from the liver — are well known.

Cannon (1927) described this pattern in some detail, arguing that this kind of high-intensity, undifferentiated arousal accompanied all emotions. Among contemporary theories the notion of undifferentiated arousal is most clearly found in Mandler's theory (Mandler, 1975). However, undifferentiated arousal also played a major role in the extraordinarily influential cognitive/physiological theory of Schachter and Singer (1962). According to this theory, undifferentiated arousal is a necessary precondition for emotion — an extremely plastic medium to be moulded by cognitive processes working in concert with the available cues from the social environment.

At the other extreme are those who argue that there are a large number of patterns of ANS activation, each associated with a different emotion (or subset of emotions). This is the traditional specificity position. Its classic statement is often attributed to James (1884), although Alexander (1950) provided an even more radical version. The specificity position fuelled a number of experimental studies in the 1950s and 1960s, all attempting to identify some of these autonomic patterns (e.g. Averill, 1969; Ax, 1953; Funkenstein, King and Drolette, 1954; Schachter, 1957; Sternbach, 1962). Despite these studies, all of which reported support for ANS specificity, the undifferentiated arousal theory, especially as formulated by Schachter and Singer (1962) and their followers, has been dominant for a great many years.

Is the ANS capable of specific action

No matter how appealing the notion of ANS specificity might be in the abstract, there would be little reason to pursue it in the laboratory if the ANS were only *capable* of producing one pattern of arousal. There is no question that the pattern of high-level sympathetic arousal described earlier is one pattern that the ANS can produce. Cannon's arguments notwithstanding, I believe there now is quite ample evidence that the ANS is capable of a number of different patterns of activation. Whether these patterns are reliably associated with different emotions remains an empirical question, but the potential is surely there. A case in support of this potential for specificity can be based on: (a) the neural structure of the ANS; (b) the stimulation neurochemistry of the ANS; and (c) empirical findings.

Structure

Although the sympathetic branch of the ANS has a great deal of ganglionic mixing, which supports a kind of diffuse unified action, the parasympathetic branch is constructed quite differently. In the parasympathetic branch there is little ganglionic mixing; rather there are a number of separate major nerves (e.g. the pelvic nerve, the vagus nerve). Organs as disparate as the stomach, the heart, the salivary glands and the tear glands are served by parasympathetic nerves, and there is little basis for asserting that these organs act in an all-or-none fashion.

There are other structural aspects of the ANS that make possible a number of different patterns of activity. The distribution of blood flow to different parts of the body is regulated primarily by the ANS through its control of vascular dilatation and constriction. This intricate system is clearly capable of a large number of different patterns of activation, redirecting blood flow from one part of the body to another to meet demands imposed by processes as disparate as exercise and digestion, as well as responding to quite localized needs such as those associated with changes in temperature and with injury.

Stimulation neurochemistry

Since norepinephrine is the major neurotransmitter at the junction between the sympathetic nerves and the organs they serve, and since norepinephrine is released into the blood stream by the adrenal medulla under conditions of sympathetic nervous system arousal, it is often asserted that this produces a diffuse and global action. Again there is an undeniable truth to this, especially at high levels of sympathetic nervous system arousal in which sufficient quantities of norepinephrine are released. However, in the case of a short-lived emotions which occur at moderate levels of intensity, it is doubtful that the amount of norepinephrine released into the blood stream from the adrenal medulla is sufficient, nor is enough time available, for it to have a significant global effect.

There is additional potential for specificity. At least two major ANS organ systems served by the sympathetic nervous system do not operate according to the 'standard' norepinephrine neurochemistry; both the sweat glands and the adrenal medulla are activated by acetylcholine instead. Thus, these systems would be unaffected by the amount of circulating norepinephrine. Finally, our understanding of the receptor neurochemistry at the typical sympathetic nervous system organ receptor site has changed considerably with the discovery that not all sympathetic organs have the same kinds of receptors. At least three kinds of receptors are now known to exist in the sympathetic nervous system (α, β_1 and β_2 receptors), each with a somewhat different stimulation chemistry. These receptor sites are not distributed uniformly

throughout the sympathetic nervous system. Although we do not know what role, if any, these different kinds of receptors play in emotional states, they do provide additional potential for differential patterns of response.

Empirical findings

Over the years there have been a number of lines of empirical research in psychophysiology that have demonstrated that the ANS is capable of different patterns of activation. Perhaps the best-known have been the studies of autonomic concomitants of cognitive–perceptual states such as orientation and defence (e.g. Sokolov, 1963) and stimulus intake and rejection (e.g. Lacey, *et al.*, 1963). In addition, there are the admittedly more controversial studies of operant conditioning of different ANS patterns in animals (e.g. Miller, 1969), which showed the potential of the ANS for producing a number of quite distinctive patterns both within and across organ systems. Human research using biofeedback could also be cited in this context, but there the evidence for differential ANS response is much weaker (e.g. Levenson and Ditto, 1981).

Discrete versus dimensional models of emotion

Contemporary research on emotion has been dominated by two quite different models of how human emotions are organized; (a) the discrete model and (b) the dimensional model. The discrete model envisions an emotional landscape consisting of well-defined singular 'primary' emotions such as fear, anger and sadness. The emphasis in research influenced by this model has traditionally been on uncovering the internal organization of these singular emotions, rather than on exploring their interconnections. The dimensional model, in contrast, envisions a multidimensional space consisting of a limited number of underlying dimensions such as negative–positive, strong–weak and active–passive, upon which are located a large number of emotional states. The emphasis in dimensional research has traditionally been on discovering the interconnections and relations among emotions.

Until quite recently the flavour of empirical studies that grew out of each of these traditions was quite different. The discrete model gave rise to work that was biological and evolutionary in flavour. Work on autonomic specificity clearly was the province of the discrete emotions model, as it attempted to uncover the autonomic differences between primary emotions such as fear and anger. The dimensional model gave rise to work that was much more cognitive and social, concerned primarily with understanding the ways in which people think about and form judgements about emotional phenomena.

Recently, dimensional models have taken a noticeable turn towards physiology. In the realm of the ANS, Winton, Putnam and Krauss (1984) have attempted to determine if different ANS responses are lawfully related to

different dimensions of emotion. Their research provides some support for the notion that the pleasantness dimension of emotion is primarily associated with heart rate, while the intensity dimension is primarily associated with skin conductance.

Interestingly, a number of CNS researchers studying specificity of brain activation in emotion have also adopted the dimension model. For example, Davidson and Fox (1982) have argued that the region of maximal cortical activation during emotion is predicted by the location of the emotion on the 'positive–negative' dimension (with positive emotional states associated with left frontal activation and negative emotional states associated with right frontal activation).

It is my belief that the most fruitful model for exploring *autonomic* specificity will prove to be a hybrid primarily on the discrete emotions model, but also taking into account the dimension of 'intensity' (see below for a more complete discussion of intensity). Given the limited number of studies to date, the inconsistency of findings and the methodological problems that have characterized much of the work on ANS specificity, there is much to be gained from pursuing separate lines of investigation within both the discrete and dimensional models. When findings have accumulated from a sufficient number of sound studies representing each model, it should then be possible to evaluate their relative usefulness

How many emotions are there?

This is a question for which no easy answer exists. For dimensional models this has not been a primary concern, since even a simple single bipolar dimension implies the possibility of a large number of different emotional states. Multidimensional models provide even greater possibilities; each point on a given dimension can be expanded along the other dimensions, resulting in an infinite number of possible emotions. While there has been some recent discussion concerning the number of dimensions that are needed to adequately map the emotion space (e.g. Smith and Ellsworth, 1985), most dimensional theorists continue to work with two or three dimensions.

For discrete models the answer to the question of how many emotions there are is much more critical, since it sets limits on the scope of investigation. It is important to realize that this answer will be different depending on which component system of emotion is being studied. In general, as the sphere of investigation moves from that which is uniquely human to that which reflects an increasingly broadly based biological heritage, the number of reliably differentiable emotions will become increasingly smaller.

The domain of written language provides an example at one extreme. To answer the question of how many emotions there are in this domain, we would need to compile a list of all words in a given language that describe an

emotional state. In contemporary languages the number of emotion terms in such a lexicon would be extremely large. Removing synonymous, applying clustering techniques and the like, would reduce the set of emotions somewhat, but the number of different emotions could easily run into the hundreds. In contrast, when one moves into the realm of the skeletal muscles such as those involved in emotional facial expression, the number of emotions which can be reliably distinguished is much smaller. Theorists differ as to the exact number, but most scientists who have studied the face hold that there are fewer than ten emotions that can reliably be distinguished on the basis of facial expression (e.g. Ekman, Friesen and Ellsworth, 1972; Izard, 1971).

In the domain of the autonomic nervous system, I believe the number of reliably distinguishable emotions will prove to be even smaller. Based on our own data, and data from those who have worked in the field before us, an optimistic but reasonably conservative prediction would be that we will ultimately be able to distinguish four negative emotions on the basis of their associated ANS activity: fear, anger, disgust and sadness. Beyond these four, only hopefully informed speculation can be offered. Under the heading of speculation, I expect that we will also be able to distinguish a positive emotional state of 'relaxed happiness' from the aforementioned four negative emotions on the basis of the *absence* of certain autonomic signs. In addition, two other emotions might ultimately be distinguished: surprise and amusement. But distinguishing these latter two emotions will probably require looking at the ANS in a somewhat different way, taking into account not only the *kind and amount* of ANS activity, but also aspects of its *timing* (e.g. speed of onset) and *duration*.

For those who embrace the notion of a boundless set of autonomically distinguishable emotions along the lines originally proposed by James and Alexander, my expectations for only a modest degree of ANS specificity may seem overly gloomy. However, as I hope to make clear in the following sections, there are still a number of extremely difficult methodological problems that must be solved before we can begin to explore systematically even the most limited set of distinctions among emotions based on the ANS.

Baseline

Among the problems involved in studying ANS activity during emotion, perhaps none is more basic than the issue of obtaining a reasonable baseline condition against which to compare emotion-related ANS changes. Establishing a baseline condition is a prerequisite for being able to make statements about the directionality of ANS changes during emotion. Thus, if one's model of anger includes a belief that heart rate accelerates during anger, the question is begged: acceleration in relationship to what prior level, obtained during what kind of prior condition? In this section I would like to discuss briefly the virtues

and weaknesses of several kinds of baseline procedures.

Resting baseline

A 'rest' period would seem at first glance to provide a highly desirable baseline condition, and many investigators have adopted a baseline procedure in which the subject is asked to do essentially nothing. In our own work using mental imagery to elicit emotions, we first instruct subjects to 'rest and empty your minds of all thoughts, feelings, and memories'. Leaving aside for a moment the question of whether a subject can actually comply with these instructions, we need to examine the pros and cons of using this kind of rest period as a baseline.

It is my belief that the best baseline or point of comparison for any biological system is one that is representative of the modal level of activation for that system, taking into account its normal range of functioning. The ANS is a slave to many masters, and unfortunately for our research, 'rest' is not a modal condition for the ANS. Research over the years has linked ANS activation to a wide range of behavioural, cognitive and perceptual states, in addition to emotional states. Emotion, in its natural occurrence, is rarely superimposed upon a prior state of 'rest'. Instead, emotion occurs most typically when the organism is in some state of prior activation. To understand how the ANS responds in emotion, we might well be better off adopting a baseline procedure that produces a moderate level of ANS activity, rather than starting with 'rest', which produces perhaps the lowest level of ANS activation that the system is capable of.

There are both methodological and theoretical virtues associated with using a baseline activity that produces a moderate level of ANS activation. From the standpoint of methodology, starting with the ANS in the middle of its range of activation opens the possibility for change in both the increase and decrease directions, without immediately running into biological floor and ceiling limits. From a theoretical viewpoint we will learn more about the capacity of emotional states to activate the ANS if we start with a moderately aroused system. To find that a state of anger increases the rate of the heart's beating compared to rest may only reflect the reality that *almost any* activity compared to rest produces cardiac acceleration. In contrast, finding that anger still produces cardiac acceleration, even when the heart rate is already elevated (as it might be by having the subject do difficult mental arithmetic), tells us something about the power of anger to recruit additional cardiovascular activity, even when the organism is already somewhat aroused. Whereas any kind of activity might increase heart rate from 'rest', it may be uniquely the province of strong negative emotions such as anger, to be able to increase heart rate from already elevated levels.

The need for an alternative to 'rest' for use as a baseline in research on ANS specificity has become increasingly apparent to me as our own work on

emotion has progressed. In the search for alternatives, some creativity will be needed to devise an appropriate baseline condition for a given emotion-eliciting task. Isomorphism may be a useful principle for devising baseline procedures. For example, when we have attempted to elicit emotions by having subjects make a set of facial muscle contractions that are prototypical for a given emotion, we have obtained a baseline by having them make a set of muscle contractions that produce a facial expression with no emotional meaning (Ekman, Levenson and Friesen, 1983). Another selection principle would be to adopt a baseline activity of theoretical interest. For example, I believe that the evolutionary meaning of positive emotions such as happiness might be to function as efficient 'undoers' of states of ANS arousal produced by certain negative emotions. To test this hypothesis a reasonable baseline condition for the investigation of ANS concomitants of happiness would be one that produces a prior state of fear, anger or sadness.

The alternative of no baseline

One way to eliminate the problem of finding an appropriate baseline is to eliminate the baseline entirely, making all comparisons between samples of emotions without regard to calculating changes from some reference point. We have been utilizing this approach in our current research to carry out subject-by-subject analyses across studies that have utilized different tasks and different baseline procedures. For example, we believe that heart rate acceleration is a component of the emotion of sadness, while heart rate deceleration is a component of disgust. A no-baseline analysis can be carried out within a given eliciting task by determining the proportion of cases in which heart rate was faster during sadness elicitation than during disgust elicitation.

There are two major disadvantages of this approach. First, we can only use data from a given subject on a given task if that subject has produced usable trials for each emotion in the comparison. Thus, if we are interested in a sadness versus disgust comparison, and a subject has an unusable sadness trial, we also lose the data from the disgust trial, no matter how good that trial was. Second, we lose the ability to describe ANS activity in terms of directional changes from some pre-emotional state. The loss of directionality, in addition to impoverishing the quality of description, exacerbates an already potentially serious confound involving the intensity of an elicited emotion (this problem will be addressed in the following section).

Finally, there is one other consideration that needs to be raised when considering ANS baselines — the stability of ANS measures over time. Although the ANS has traditionally been considered to be a 'centre-seeking' or homeostatic system, the centre point can change over time. Beyond very real, significant, and all-too-common problems associated with not allowing adequate time for subjects to adapt to the experimental environment, there may be

slow linear trends in individual response channels over time. The existence of these trends requires some attempt to obtain repeated baseline readings so that changes are always being computed in relationship to a contemporary basal level. Among the traditional ANS measures, heart rate is probably the most likely to return to a stable central setting. Measures of skin conductance or skin resistance level are more troublesome (especially if an electrolyte is used that changes its own conductivity over time). In my experience, peripheral measures of skin temperature are the most vulnerable to these problems; even under well-controlled conditions of external room temperature, subjects often show slow linear drifts in basal peripheral temperature throughout the course of an experiment.

Lest the reader think that the problem of baseline is overly academic, the baseline procedures utilized in several of the classic studies of ANS specificity demonstrate a number of different baseline problems. For example, Sternbach (1962) had children view a 1-hour film ('Bambi') and identify the scenes they found to be most 'sad', 'scary', etc. The ANS data that occurred during these scenes were then extracted. The baseline period used for comparison, however, was extracted from the 30–60 seconds of film that preceded the selected scene. Thus, the content of the various pre-scene baselines was in no ways comparable, rendering the data for each emotion — which were reported in terms of changes from baseline — essentially meaningless. Another example comes from Schachter and Singer (1962), where a measure of heart rate was obtained following each of two 20-minute emotion-elicitation procedures. The baseline obtained in that study was a single heart rate reading taken near the beginning of the experiment, just prior to subjects receiving an injection of either epinephrine or placebo. Thus, in this study the baseline was poorly chosen both in terms of time (i.e. too far from the emotion elicitation) and context (i.e. waiting for an injection is not a very modal state for the organism).

Intensity

Although a regular feature of research in the dimensional tradition, intensity has, with few exceptions (e.g. Roberts and Weerts, 1982), been completely ignored in studies of ANS specificity during discrete emotions. Assuming that any discrete emotion such as anger can exist over a range of different intensity levels, the issues are quite basic. If a given set of ANS changes occurs at a moderate level of anger, what will happen when anger occurs at a high level of intensity? Will the changes be exaggerated, or will quite different changes occur? At a low level of emotional intensity, similar questions can be raised. Will the changes be attenuated, nonexistent, or different from those that occur at moderate levels?

Before turning to a consideration of this issue it will be useful to deal briefly with emotions that occur at the absolute extremes of intensity. Assuming for a

moment that there are emotions that are associated with distinctive patterns of ANS activity, I believe that there exists a level of intensity for such emotions sufficiently low that no discernible ANS activation will occur. Similarly, it is likely that there is level of intensity so high that the configuration of ANS activation normally associated with the emotion will be distorted by natural biological ceilings and floors that are reached, by neurohormonal factors that alter ANS responses, and by compensatory mechanisms that will act to protect the organism from permanent damage. And finally, there may be temporal conditions such as those involving repeated or prolonged elicitations under which habituation and fatigue will distort ANS patterns. In terms of adaptation, emotions may function most efficiently at moderate levels of intensity, during single elicitations and for relatively short periods of time. And it may be under these optimal conditions that ANS specificity will be most pronounced.

Hypothetical models of intensity

Because so little experimental work has been done with intensity it may be useful to develop some hypothetical models of how emotional intensity might be reflected in the ANS. Two such models can be termed the 'multiplicative' model and the 'additive' model.

Let us start with some data from our own laboratory from a study in which voluntary contraction of facial muscles was used to produce prototypical emotional facial expressions (Ekman, Levenson and Friesen, 1983). The intensity of self-reported emotion produced by this task and the magnitude of ANS changes are quite moderate. The actual heart rate changes for four negative emotions using a baseline consisting of a non-emotional facial expression are plotted in the left-most chart of Figure 2.1. Also in this figure are two extrapolations of these data depicting results that *might* accompany higher levels of emotional intensity. The centre chart illustrates a 'multiplicative' model in which the ANS changes that occur at one level of intensity are multiplied by a factor reflecting the change in intensity. Thus if the intensity of the elicited emotions were presumed to double, a heart rate increase of 8 bpm at the original level of anger intensity would become a heart rate increase of 16 bpm at the higher intensity; while a heart rate decrease of 0.5 bpm during moderate disgust would become a decrease of 1 bpm during doubly intense disgust. In the multiplicative model any directional differences between emotions become even more pronounced at higher levels of intensity.

The right-most chart in Figure 2.1 illustrates an 'additive' model in which increased intensity carries with it a constant increase in heart rate regardless of the emotion that is intensified. Here the assumption is that a doubling of the intensity of emotion will add an additional 8 bpm of heart rate increase regardless of the emotion. In this case the heart rate increase associated with intense anger again becomes 16 bpm (8 bpm + 8 bpm), but now the heart rate

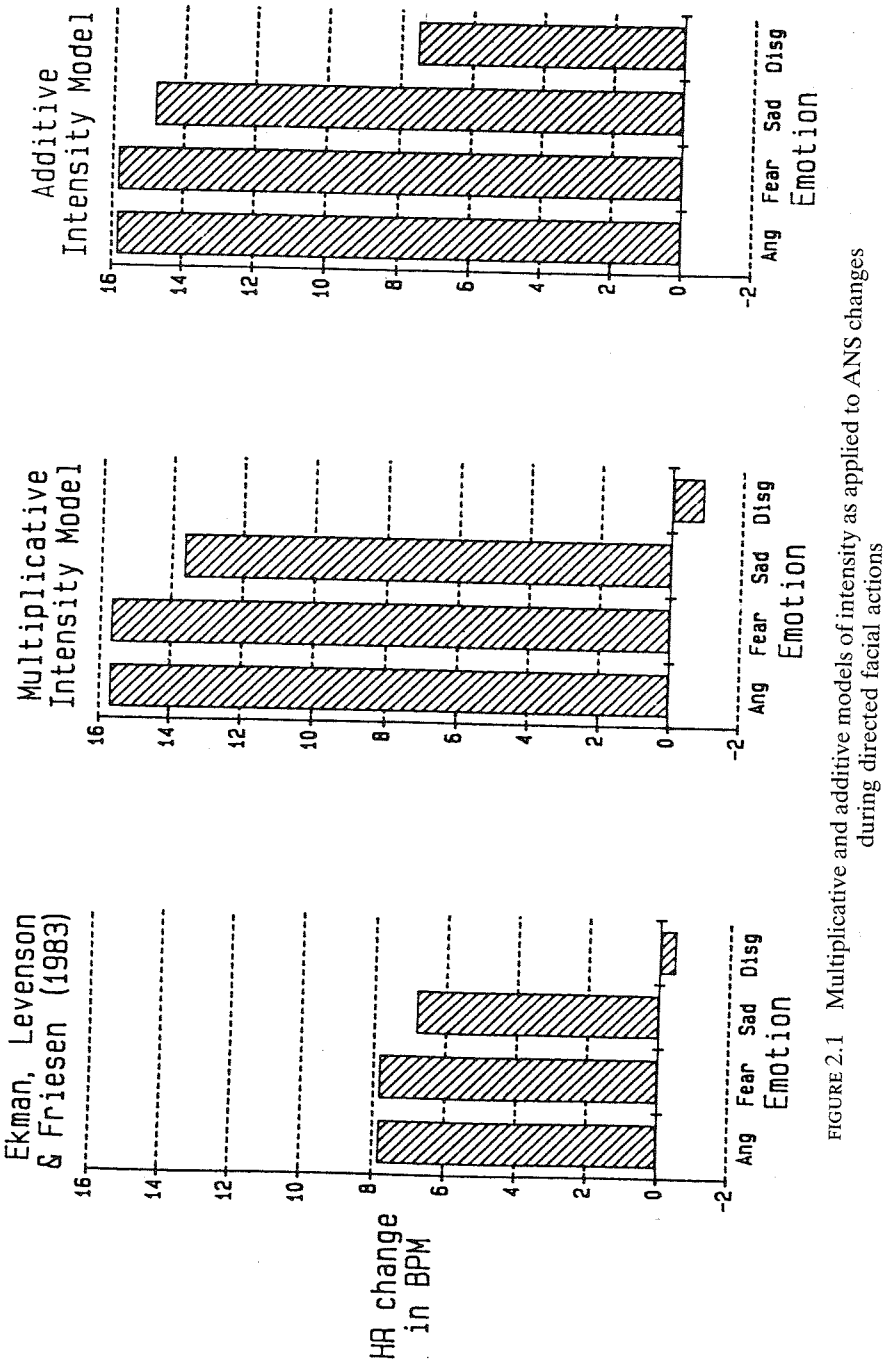

FIGURE 2.1 Multiplicative and additive models of intensity as applied to ANS changes during directed facial actions

change during intense disgust is an increase of 7.5 bpm (− 0.5 bpm + 8 bpm). In the additive model at the higher level of intensity the relative configuration among the emotions is maintained (i.e. heart rate is still faster in anger, fear and sadness than in disgust), but now heart rate actually increases in all negative emotions.

Several things should be noted before leaving this discussion of intensity. First, for those who would like to be able to utilize ANS changes as a way of reliably identifying emotional states, only the multiplicative model holds out much hope. Referring again to Figure 2.1, under the additive model a heart rate increase of 7.5 bpm could just as easily indicate moderate levels of sadness as high levels of disgust. Second, the only way to give the multiplicative model a fair test is to include some sort of reference baseline, for without this there will be no way to determine directionality of change, a necessity for this model.

We have just started to explore this intensity dimension in our own work, and it is premature to speculate about which of these models (or some other model) will ultimately be supported. One possibility that is emerging, however, is that intensity could well be reflected in different ways in different ANS response systems.

Timing

The temporal dimension of emotion is a matter of critical importance both for emotion theory and, more practically, for the design and evaluation of research on ANS specificity. In its simplest form the theoretical issue reduces to several questions: (a) how long does an emotion last?; (b) at what stage or stages during the emotion process does the autonomic component occur? At the practical level of methodology these questions become matters of knowing which moments in the ongoing stream of behaviour mark the beginning and the end of an 'emotion', and determining how to use these moments to guide our extraction of the concomitant autonomic responses from the ongoing stream of autonomic activity.

The question of how long an emotion lasts is an intriguing one. Ekman (1984) has provided a helpful discussion of this issue that locates 'emotion' on a temporal dimension between 'reflexes' (which are shorter in duration than emotions) and 'moods' (which are longer in duration than emotions). Although the precise location of the lines of demarcation between reflex, emotion and mood are open to debate, Ekman provides some useful guidelines. For example, the 'startle', a highly patterned set of facial movements that starts within 100 milliseconds after an unexpected gun shot and lasts for less than 500 milliseconds (Ekman, Friesen and Simons, 1985), could be viewed as a reflex — too short-lived to be an emotion. Similarly, the state of a person who has been feeling hostile for the greater part of a morning can be viewed as a mood — too long-lived to be an emotion. How long does a genuine emotion last? Ekman

(1984) suggests that it typically lasts between ½ and 4 seconds.

Shifts between emotions: facial expression and subjective experience

Before accepting as fact that an 'emotion' typically lasts for less than 4 seconds, we need to consider the various component systems that are involved. In the case of facial expression this seems quite reasonable. The emotional facial expressions that can be observed in the laboratory are quite brief and fleeting, and casual observation of facial expressions in natural settings will support the same conclusion.

Our experimental work studying marital interaction throws some light on the issue of how quickly the subjective experience of emotion can shift. In this work (Levenson and Gottman, 1983), married couples engage in a 15-min discussion about a problem area in their marriage while psychophysiological measures are obtained. Several days later each spouse returns to the laboratory separately to view the videotape of the interaction. As they watch the tape they are asked to indicate how they felt during the interaction using a rating dial device that traverses a nine-point scale between 'very negative', 'neutral', and 'very positive'. Subjects are asked to adjust the dial as often as needed, so that it always indicates how they felt. It would be more useful for the present purposes if these subjects had been asked to provide ratings in terms of discrete emotions, but these dimensional ratings provide some indication of how quickly feelings can change during a social interaction. In Figure 2.2. 10 minutes of rating dial data from a typical subject (a husband viewing the videotape of the discussion of a marital problem) are portrayed. To match these data better to the duration of a typical emotion in the face, 5-second averages were used to create this figure. Two points should be noted. First, the subjective rating of emotion along this single dimension is continually shifting and quite complex, spanning the full range of possible ratings. Second, adjacent 5-second periods can be extremely different in terms of their affective rating — sudden changes along the positive–negative dimension are quite possible.

ANS activity: the critical problem of temporal matching

If emotions can be as brief, and can shift as rapidly, as indicated in the preceding discussion of facial expression and subjective report, then the implications for autonomic measurement are profound. Every effort must be made to carefully match ANS measurement to the brief occurrence of these emotions. It is very easy to make two kinds of mistakes. First, we might measure at the wrong time and thus miss the emotion. Second, we might compute averages across overly long measurement periods which can include a number of different emotions, as well as periods of no emotion. In the latter

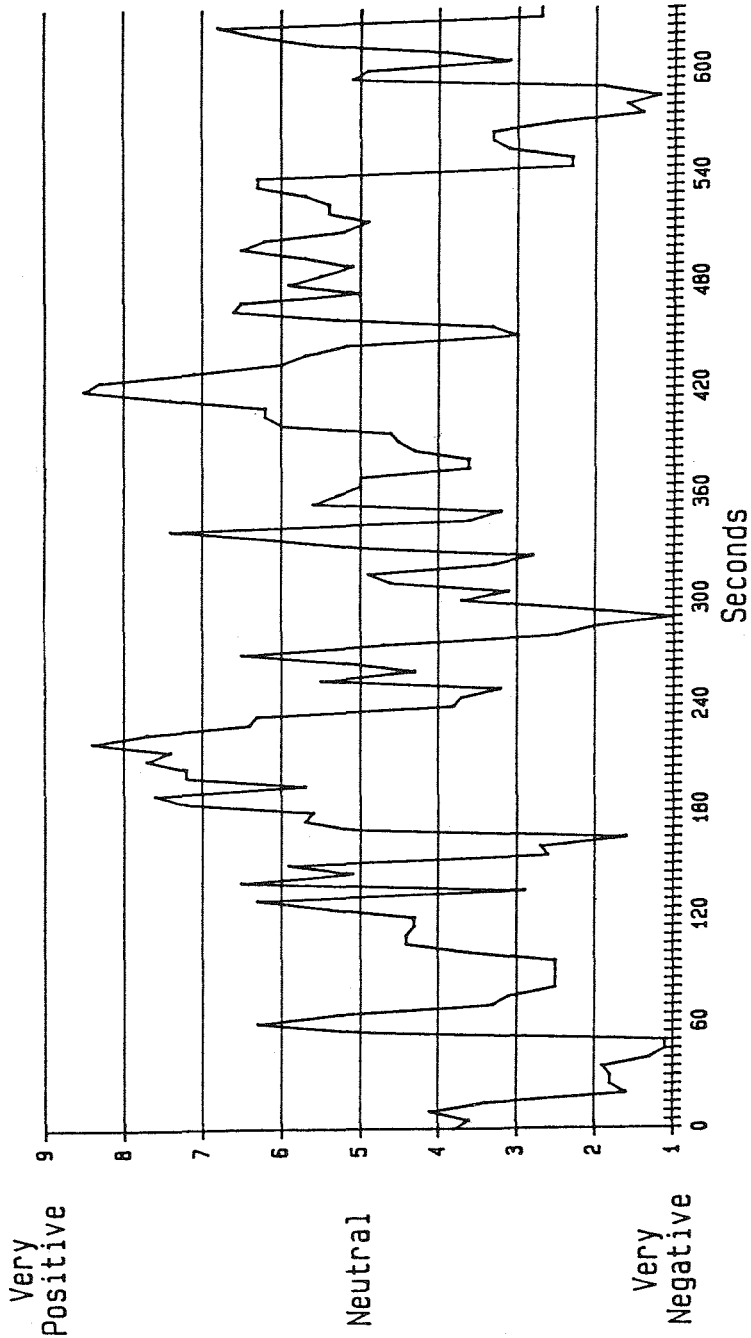

FIGURE 2.2 Rapidity of change in self-report rating of emotion during marital interaction

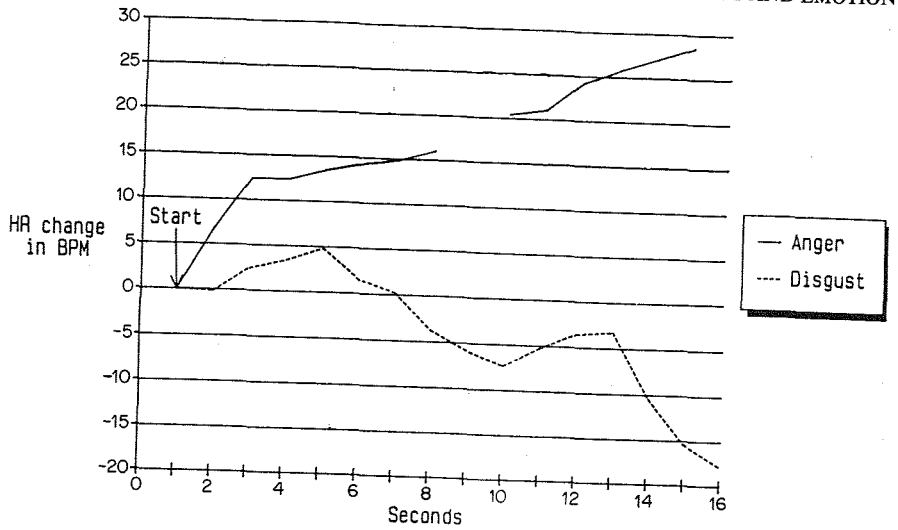

FIGURE 2.3 Rapidity of ANS changes during high-intensity emotion

case, ANS patterning associated with a single emotion could easily be obscured by the averaging procedure.

Unfortunately we know far less about the duration of ANS activity in an emotion than we do about any other component. Several commonly held notions have had a chilling effect on systematic investigation of the temporal course of ANS activity in emotion. First, there is the notion that the ANS is a very slow-acting system. Of course compared to the CNS, and to the muscles of the face, this is true. But the question really is: How fast is fast enough? For emotion, what is an acceptable latency between the initial neural impulse and the commencement of activity in the target organ? Certainly autonomic drive on the heart can cause a sizeable change in the heart's rate and force of contraction within a single heart beat (which would typically involve less than 1 second).

An example of this speed of onset and acceleration can be seen in Figure 2.3. This figure presents 15 seconds of heart rate data for a subject we have worked with who is highly skilled at producing emotions on request, and who can sustain these emotions for unusually long periods of time. For the emotion of anger (solid line), an acceleration of 5 bpm occurs within the first second after the subject was asked to 'become angry', and by the end of the next second an acceleration of 10 bpm had occurred. For the emotion of disgust (dashed line), the heart rate change is both slower in onset and more complex. For the first second following the instruction to 'become disgusted' the heart rate remains unchanged, then it accelerates 5 bpm over the next 3 seconds, before it begins a deceleration which reaches over 15 bpm by the end of 15 seconds. Although such data are certainly not conclusive, they are illustrative of the fact that the

latency of onset for ANS activity can be quite short, and the change can be quite rapid.

Another quite different notion that has impeded work on the temporal course of ANS activation is the belief that, once activated, the ANS continues to remain activated for too long a time to be able to track rapid changes in emotion. One source for this notion is undoubtedly the experience associated with extremely intense negative emotions of becoming aware of ANS activation (e.g. pounding, racing heart) which continues long after the eliciting event has passed. This kind of autonomic 'overhang' is a real phenomenon, probably resulting from the high levels of sympathetic hormones (epinephrine and norepinephrine) that are released into the blood stream from the sympathetic nerves and the adrenal medulla during intense emotional experiences. These hormones can maintain sympathetic nervous system activation at heightened levels for relatively long periods until they are eventually deactivated.

Without minimizing the importance of high-intensity emotion, we need to find out more about the temporal dimension of the *offset* of ANS activity brought about by *moderate* levels of emotion as well. Perhaps there will still be some overhang at moderate levels of emotion, but there is no compelling reason to think that the ANS component of an emotion could not onset and offset within a 10-second window encompassing a single emotion. And the onset–offset interval could be even shorter for certain ANS functions such as heart rate, in which the action of the parasympathetic branch of the ANS has the opposite effect from the action of the sympathetic branch. For such dually innervated organs a natural braking mechanism is available for restoring the ANS function back to its prestimulus levels.

The research literature on ANS specificity has numerous examples in which ANS responses have been imprecisely matched to the occurrence of emotions. Funkenstein, King and Drolette (1954) used a 2½-minute procedure to elicit emotions, but the ANS data that were analysed were obtained *after* the procedure was over. Similarly, Schachter and Singer (1962) utilized complex 20-minute elicitation procedures to produce euphoria and anger, but their ANS data (a single measure of heart rate) were obtained *after* the procedure had ended.

Verification of emotion

Failure to verify adequately *what* emotion subjects are feeling, and *when* they are feeling it, constitutes probably the most pervasive failure in the existing research on ANS specificity. Adequate verification is admittedly an extremely difficult task, but it is necessary before we can begin to understand the role of the ANS in different emotions.

Several factors contribute to the need for verification. I have already discussed at some length the problems associated with the temporal dimension

of emotion and the resultant need to locate precisely when an emotion occurs, so that ANS measurement can be matched with the occurrence of the emotion. This task is critical because the ANS at any moment can be functioning in the service of any of several masters, ranging from the relatively mundane (such as responding to changes in posture) to the extremely profound (such as responding to a threat to survival). What we need to find are those moments when the ANS is in the service of emotion; for our purposes this is the signal — all else must be considered noise.

In the natural environment any emotion can be brought forth at any time; hardly an ideal situation for obtaining tight experimental control. Thus when we attempt to study emotion in the laboratory our first order of business is to recapture some degree of control over the eliciting stimulus. This has often led to the selection of stimuli selected from the psychological house of horrors such as electric shock, loud noise, buckets of freezing water, slides and films of assorted medical, industrial and anthropological gore. Regardless of how carefully we control these stimuli, our efforts are destined to be frustrated by a basic tenet of research on emotional specificity; namely, *that the starting point for any analysis must be the occurrence of a given emotion, and not the administration of a given stimulus*. In emotion research we are not really interested in the ANS response to a precisely controlled electric shock, but rather the ANS response that occurs during the emotion of fear that *might* be produced by that shock.

The unfortunate reality is that there is no stimulus that will produce the same emotion, in all subjects, at all times. For this reason it is pointless to pursue research on ANS specificity in emotion without employing the best possible means to sift through all of the possible behaviours provoked by our elegant manipulations, to find those moments of 'emotion', and to identify these moments as to *time* and *type*.

Since the dependent measures in specificity research are responses of the autonomic nervous system, verification — if it is to avoid circularity — must make use of some other response system for which translation rules are available linking responses in that system to emotional states. Given the present state of knowledge this typically means verifying on the basis of self-report or facial expressions or both. Each of these methods of verification has its own virtues and potential pitfalls.

Self-report and some alternatives

Although often a magnet for criticism, self-report measures — carefully and skilfully used — provide one of the most powerful methods of verification available for research on ANS specificity. Self-report has the virtues of availability and economy, since it is accessible to all researchers and does not require extensive instrumentation, training or scoring time. Of course, self-report leaves the ultimate responsibility for identifying emotional states in the hands of the subjects, whose ability to accurately identify feelings will vary from individual to individual. The quality of self-report will also vary greatly as a function of the difficulty of the required discrimination, and our own skills in asking the right questions, in the best manner, and at the appropriate time.

The paper-and-pencil inventory is only one of several methods available for obtaining emotional self-report. Richard Davidson and Paul Ekman, in their ongoing work on EEG specificity, obtain verbal reports of discrete emotions repeatedly during their experiments by having subjects rate the intensity of a number of discrete emotion terms presented on slides.

Rating dials are another option. The video recall procedure that John Gottman and I have used in our work on marital interaction described previously (Levenson and Gottman, 1983) provides a fairly unobtrusive way to obtain continuous self-report ratings on a single dimension. Although this method appears to have reasonable validity (Gottman and Levenson, 1985), it rests on the assumption that viewing a videotape will be a sufficient stimulus to recapture past emotional experiences. This assumption would be difficult to support in contexts other than intimate social interaction. In my own work with film viewing and laboratory stressors I have often used rating dials to obtain a continuous emotional rating on a single dimension throughout the actual experiment (e.g. Levenson *et al.*, 1980). This procedure could be adapted to have subjects provide continuous ratings on one or two discrete emotions.

Finally, although contemporary experimentalists tend not to favour it, there is the interview procedure. A skilled interviewer, experimentally blind, working with a subject immediately following an experimental manipulation, might be able to obtain a more veridical self-report of emotions than could be obtained using a sterile sheet of paper. For those interested in learning about the nuances of emotional interviewing the interview techniques used by phenomenological psychologists to obtain more precise emotional self-reports can be extremely helpful (e.g. de Rivera, 1981).

Common procedural errors with self-report

Effective use of self-report verification methods requires avoiding two common procedural errors. The first is the use of self-report measures that produce emotion ratings that do not match up with the emotions under study. Thus, if one is interested in comparing ANS differences between discrete emotions such as anger and amusement, it is important to use a self-report measure that produce scores for discrete emotions rather than ratings on emotional dimensions; and to use a measure that produces scores for 'anger' and 'amusement' rather than for 'hostility' and 'joy'. Finding equivalencies between emotional terms in a language is a painstaking task that often reveals non-intuitive results; thus it is best not to assume equivalencies on an *a priori* basis.

The second basic error is one of timing. The self-report procedure should be designed so that the rating is obtained as close in time to the actual emotional experience as possible, since retrospective emotional recall may be much less accurate than more immediate reports. In addition, the procedure should focus the subject's rating on a brief period of time to maximize the likelihood that the rating will not include a series of different emotional and non-emotional events. Studies in which subjects endure emotionally complex and lengthy manipulations and then — after all of the fireworks are over — are asked to provide a one-shot emotional rating for verification are misguided (making the same basic temporal error as in studies in which ANS measurement is obtained long after any elicited emotions have subsided).

Examples of long delays prior to obtaining self-report can readily be found in the existing literature. For example, in Ax's (1953) study of fear and anger, subjects were not asked about the feelings that had occurred during a complex 5-minute elicitation procedure until at least 30 minutes later. During the intervening period the subject had been exposed to another complex elicitation procedure for a second emotion. Schwartz, Weinberger and Singer (1981) waited 7 minutes after each of their imagery trials to ask subjects how they felt; this waiting period included a 1-minute period of vigorous physical exercise.

One other puzzling practice is sometimes followed with self-report data. Subjects are questioned about their feelings, and these data are used to verify that the experimental manipulations were effective by showing, for example, that subjects exposed to an anger manipulation reported more anger than those exposed to a sadness manipulation. However, when physiological data are analysed, data from *all* subjects in a given condition are included. In such cases ANS changes associated with anger could be based on an odd assortment of subjects, including some who actually reported feeling angry, as well as others who reported feeling nothing or some emotion other than anger. In such studies a good opportunity to utilize verification data to 'purify' samples is lost.

Facial expressions

Using facial expressions to verify subjects' emotional states has considerable appeal, especially since it can be done unobtrusively and continuously from a videotape recording of the subject's face obtained during an experimental procedure. In our work on emotion we routinely obtain these kinds of videotapes and time code them in such a way that we can precisely match the concomitant ANS activity that occurs during an emotional facial event. From the perspective of avoiding the vagaries associated with self-report, verification using facial expressions represents a major methodological breakthrough for research on ANS specificity. Despite the enthusiasm that I have for facial measurement, there are several caveats that should be raised.

First and foremost, there is the *sufficiency* issue. The use of facial expression

to verify or detect emotional moments rests on the assumption that what we see on a subject's face is a true indication of what the subject is feeling. Despite some fairly good evidence in support of this assumption for the primary emotions, a number of legitimate questions can be raised about accepting the occurrence of an emotional facial expression as a sufficient basis for inferring an internal emotional state.

There is also the *necessity* issue. It may be overly conservative to postulate that the occurrence of an emotional facial expression is necessary for the presumption of emotion. Few would argue that the absence of the complete prototypical expression for a given emotion guarantees that the subject is not feeling that emotion. Even the most radical advocate of the centrality of facial expression in emotion would admit the possibility that, at least at lower intensities, we can feel a primary emotion and produce only subvisible changes in the face, or produce visible changes that are ambiguous, leaving out elements of the full complement of muscle contractions that are prototypic for the emotion. In fact, with the possible exceptions of happiness and disgust, it is unusual to see the full-facial prototypes of primary emotions in response to most laboratory manipulations.

The experimental situation can also play a major role in altering the threshold for the appearance of emotional facial expression, as well as altering the kinds of expressions shown by subjects in the laboratory. Ekman and Friesen (1969) have written extensively about the role of a culture's display rules, and how they can alter the facial behaviour seen in response to an experimental manipulation.

Finally, the *costs* of facial measurement are high both in terms of the level of training needed by coders, and the time required to adequately code the face from videotape. For precise muscle-by-muscle measurement it can easily take 30 minutes to code a minute of facial behaviour; and once the coding has been completed one needs to apply a set of translation rules to the codes to extract their emotional meaning. Fortunately for the field, there are several well-developed systems for doing this kind of facial measurement (the two most prominent systems are associated with Ekman and Friesen, 1978, and with Izard, 1979; see Ekman, 1982 for a comparison of several available systems).

ANS activity as an independent criterion for detecting the occurrence of emotion

Although it would be circular to look to the ANS as a means of verifying the occurrence of emotion in research on ANS specificity, it is interesting to speculate on the possibilities of using the ANS as an indicator of emotion. Should we ever reach a point in this research area where we have identified ANS patterns for a number of emotions, we might be able to use the ANS as an independent criterion for emotion. This would be a potentially invaluable tool

for studying the interplay among the other emotional components, especially facial expression and subjective awareness.

The notion of using the ANS in this manner is a simple one. For example, if we believe that the ANS pattern for fear consists of an acceleration of heart rate of at least 5 bpm, accompanied by a decrease in finger temperature of at least 0.5 °F, then we could scan a subject's ANS record from an experiment to find instances that match this pattern. At the very least we could pose questions of conditional probability. For example, given the occurrence of this 'fear' pattern in the ANS, what is the probability of the subject self-reporting fear, of the face showing an expression of fear, of the environmental event being conducive to fear? If these probabilities are high in 'normal' subjects, there might be considerable clinical utility in using ANS patterns to explore the effects of such psychodynamic constructs as repression, during which we would expect a discrepancy to appear between the occurrence of an emotion (as indicated by the ANS) and its articulation in expression and subjective awareness.

A question of importance

The primary question concerning ANS specificity has always been: Does it exist? However, an equally important question that should be raised is: If specificity exists, does it matter? Autonomic differences between emotions could well exist but be of little consequence, merely representing vestiges of an earlier biological organization that is no longer critical for the emotional life of the human species. My position on this second question is that ANS specificity does matter, and I would like to conclude this chapter by discussing briefly why this may be so.

Initiation and completion of emotion

There are those who would assert that the essence of emotion only emerges in its total *gestalt*; if activation of any one of the major component systems is missing — whether it is facial expression, cognition, or ANS arousal — what is left is not quite 'emotion'. The debaters of these issues are clearly concerned with the necessary conditions for the occurrence of emotion. Arguments over sufficient conditions can also be raised that pose the question of whether activation of any single component system — expression, cognition, or ANS arousal — is by itself an emotion. A third, related argument concerns initiation — the capacity of each component system to recruit the other component systems to produce a full-blown emotion.

It would be hard to argue that ANS arousal is a sufficient condition for emotion, since ANS arousal accompanies many potentially non-emotional activities such as exercise, postural change and digestion. Of course, it is

possible that the *patterns* of ANS arousal that accompany emotions are unique, never duplicated in non-emotional states. In my view the assertion of sufficiency of ANS arousal for emotion is as tenous as the assertion of sufficiency for facial expression or for cognition. On the other hand, I do believe that a full-blown emotion can be *initiated* by activation of either the ANS or the face or cognition. However, progression to the completion of the full emotion will require activation of the collateral systems as well as adequate contextual support from the environment. In this viewpoint the ANS is neither king nor pawn; it plays a central role, but not an exclusive one.

Colouring the subjective experience

Autonomic differences between emotions could help determine a number of central features of the subjective experience of each emotion, even contributing to the ultimate cognitive labelling of the emotional state. I believe that there is not yet sufficient evidence available to decide the issue of whether or not physiological changes (whether they occur in the ANS, in the muscles of the face, or in the large skeletal muscles) play a significant role in shaping the phenomenological experience of emotion. For the ANS to play such a role it is necessary that some mechanism exist for providing feedback of ANS changes into the central nervous system structures that are responsible for producing the subjective experience. That such proprioceptive mechanisms exist in the ANS cannot be questioned; but whether they are sensitive enough to provide the kind of information that influences cognition is less certain. In addition, there remains the elusive question of the mechanisms by which any available proprioceptive information from the ANS would be utilized. I believe that we can become consciously 'aware' of the heart's pounding, and of the stomach's churning. However, the influence of ANS changes on subjective emotional experience are likely to be much more subtle than depicted in the 'I churn; therefore I feel' model, in which the proprioceptive evidence is weighed and mulled over before the emotional verdict is rendered.

Evolution, adaptation, health and disease

The prominence afforded to human cognition by contemporary psychology should not be allowed to obscure the evolutionary importance of autonomic specificity. If different patterns of ANS activation are found to exist for different emotions, and even if these patterns are found to have *no* discernible impact on cognition, ANS specificity could still have profound survival value for an organism forced to respond efficiently and appropriately to a number of different environmental demands.

This is the most basic argument in support of the critical importance of ANS specificity. When faced with a situation which requires sudden behavioural

adaptation the ANS has to quickly assume the proper configuration to support that behaviour. 'Fight' and 'flight' are not the same behaviours. They involve different groups of skeletal muscles, and require quite different kinds of autonomic support. If there is an ANS pattern associated with anger then it should be one which is supportive of the behaviour of 'fighting'; if there is an ANS pattern associated with fear then it should support 'fleeing'. A pattern of gastrointestinal activation associated with disgust would not be supportive of these kinds of behavioural adaptations. Emotion provides a mechanism by which behaviour, facial expression and the *appropriate ANS support* can be quickly matched to the immediate environmental demands. The capacity of the ANS for supporting a limited number of primary emotional/behavioural pairings is the centrepiece of its evolutionary value in emotion.

In the contemporary era we are usually quite insulated from the kinds of stark environmental forces that impinged on earlier man, yet there is no reason to believe that our emotional lives are any less vivid. We still can experience extremely strong emotions such as anger accompanied by the full complement of ANS activation, but now these emotions occur in a social and psychological context that often inhibits the expression of the natural behavioural concomitants. An emotionally aroused ANS, assuming a specific pattern of activation, under conditions of behavioural restraint, may have become the modal emotional condition. If this is so, then ANS specificity may become increasingly important in helping us understand the links between specific emotions and processes of health, immune function and disease.

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CHAPTER 3

Neuroendocrine measures of stress

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Abstract: Stress is a 'whole body' process involving central and peripheral nervous system changes and a variety of psychological and physiological responses. Endocrine activity, particularly catecholamine and corticosteroid response, has been established as a reliable marker of stress. The importance of endocrine activity in stress and health changes is discussed.

'Stress' is probably one of the most confusing and difficult terms to define in the biomedical and behavioural sciences. It has become a catchall explanation for many physical and mental complaints having no known pathogenic or psychological aetiology. Researchers conceptualize stress as both a psychological and physical precursor to illness, as well as a state that accounts for transient behaviour and biological change. In general the concept has acquired a negative connotation and is often associated with environmental circumstances which disproportionately tax an individual's resources because they are beyond what is typically experienced by the person. Stress is defined in this chapter as a complex of emotional cognitive, behavioural and biological responses to the threat of being harmed, to strong environmental demand or to the loss of something valued. Stress refers to a continually changing process of relationships between people and their environments; it is not simply a stimulus or a response, but rather a combination of the two (Lazarus and Folkman, 1984). As a result, stress cannot be investigated from a restricted perspective. No one discipline will unravel the complexities associated with the phenomenon of stress.

This chapter is primarily concerned with demonstrating the usefulness of incorporating neuroendocrine measures in the study of stress. It should not be inferred, however, that biochemical markers define the stress response or are more important than any other measure of stress. What will become apparent