Chapter 3
THE SYMPATHOVAGAL BALANCE
EXPLORED IN THE FREQUENCY DOMAIN

The neural regulation of circulatory function is mainly effected through the interplay of the sympathetic and vagal outflows, which are tonically and phasically modulated by means of the interaction of at least three major factors: central command (or central integration), peripheral inhibitory reflex mechanisms (with negative feedback characteristics), and peripheral excitatory reflex mechanisms (with positive feedback characteristics) (Figure 1).

To study this whole interplay only through the action of single reflexes appears an unsound illusion, since the fragmented pieces of knowledge are difficult if not impossible to reassemble into a unitary conception. Simplified but general hypotheses might be necessary. In this respect the sympathovagal interaction might furnish an appealing conceptual model.

The sympathovagal balance

In most physiological conditions, the activation of either sympathetic or vagal outflow is accompanied by the inhibition of the other (hence the concept of balance, as a horizontal beam pivoted at its center) (Malliani 1999a; Malliani et al. 1991a, 1998). This is true for reflexes arising not only predominantly from the arterial baroreceptive areas but also from the heart. For instance, we have already seen in Chapter 1 that the stimulation of cardiac sympathetic afferents induces reflex sympathetic excitation and vagal inhibition, whereas the opposite effect is elicited by stimulating cardiac vagal afferents (Schwartz et al. 1973). This reciprocal reflex organization, alluding to a synergistic design, seems instrumental to the fact that sympathetic excitation and simultaneous vagal inhibition, or
viceversa, are both presumed to contribute to the increase or decrease of cardiac performance to implement various behaviors.

The schema of Figure 1 has thus been slightly modified into a version (Figure 16) in which the central and peripheral mechanisms do not regulate a single autonomic outflow but rather a complex interaction exemplified by *sympathovagal balance*.

![Figure 16: Schema of opposing feedback mechanisms that, in addition to central integration, regulate the state of sympathovagal balance. See text and Appendix. (From Malliani et al. 1991a, with permission).](image)

It is the purpose of this Chapter to demonstrate that this interaction can be broadly explored by assessing cardiovascular rhythmicity with an appropriate methodology. In fact, variable phenomena such as heart period and arterial blood pressure can be described not only as a function of time (i.e. in the *time domain*), but also as the sum of elementary oscillatory components, defined by their frequency and amplitude (i.e. in the *frequency domain*).

**Neural and cardiovascular rhythms as markers of functional states**

Rhythmicity is an intrinsic property of the nervous system. Various rhythms can be markers of normal events, such as wakefulness or sleep, and of abnormal conditions, such as epilepsy. However, a rhythm is rarely unequivocally linked with a function: thus an atropinized cat can walk around with an