COMPARISON OF PLETHYSMOGRAMS TAKEN FROM FINGER AND PINNA DURING ANAESTHESIA

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In the preceding paper it was noted that photo-electric plethysmography could be used to monitor several aspects of the circulation (Dorlas and Nijboer, 1985). During anaesthesia the plethysmogram is obtained either from the cutaneous vascular bed of the digits, where the reflection method is used, or from the ear, where the transmission method is the obvious choice. However, when reviewing data from 500 patients in whom plethysmograms were recorded simultaneously from finger and ear during anaesthesia, we observed a marked difference in the responses to various stimuli.

In the first instance, one might be inclined to ascribe the differences to the use of the reflection and transmission methods, respectively. However, this possibility was excluded by a previous study which demonstrated no difference in the light/voltage relationship, when applied to the finger or ear (Nijboer, Dorlas and Mahieu, 1981). Therefore, the difference in response must result from factors which control the size of the blood volume pulses ($AV$) in finger and pinna, these being the variations in intravascular pressure ($AP$) and the distensibility ($D$) of the vascular walls. The relationship is given by Burton (1972) as $AV = AP.D$.

The peripheral pulse pressure in this formula is derived from the pressure in the root of the aorta, so that it can hardly account for the difference in response between fingers and ear. Consequently, we believe that the difference in response has to be attributed mainly to a difference in distensibility of the vascular walls. Since the distensibility of the arterial vascular bed depends chiefly on the tone of vascular smooth muscle, it will be controlled primarily by the autonomic nervous system (Burton, 1954).

**RESPONSES IN FINGER PLETHYSMOGRAM**

Figure 1 depicts plethysmograms (Honeywell Philips peripheral pulse module, described in the accompanying article (Dorlas and Nijboer, 1985)) from finger and pinna recorded simultaneously at the beginning of an anaesthetic.

**SUMMARY**

Pulse plethysmograms from finger and pinna were recorded simultaneously during anaesthesia, and marked differences in their response to various stimuli recorded. The differences have been illustrated by a number of examples.

**On induction** the most striking phenomenon was the marked increase in amplitude which occurred in the finger plethysmogram and which did not occur in the ear plethysmogram. This increase in "finger" amplitude (temporarily interrupted by intubation) has been recorded in 95% of inductions, and is independent of the induction agent used. This effect was...
also reported by Strandness and colleagues (1964), Otteni, Sauvage and Gauthier-Lafaye (1970), and Johnstone (1974a). Because pulse pressure does not increase, the increase in finger amplitude is attributed to a decrease in vascular smooth muscle tone,—that is, vasodilatation. Since this vasodilatation is found particularly in the finger, where the walls of the cutaneous vessels are innervated by α-adrenoceptors (Goodman and Gillman, 1965), there must be a loss of sympathetic tone induced centrally by the suppression of psychic stimuli. Therefore, it is called an “induction sympatholysis”.

Intubation, on the other hand, produced a profound decrease in amplitude, which was also observed principally in the finger plethysmogram. Since this decrease in amplitude was not accompanied by a decrease in pulse pressure, it had to be caused by constriction of vascular smooth muscle. This vasoconstriction shows that, although the patient was anaesthetized, the reactivity of the finger vessels to stressful stimuli was preserved, and predominated over the effect of pulse pressure. Furthermore, it may be noted that, initially, the plethysmograms showed small rhythmic and apparently spontaneous fluctuations: these are characteristic of the awake state (Burch, Cohn and Neumann, 1942) and always disappear during the induction of anaesthesia (Dorlas and Nijboer, 1985). Following intubation of the trachea, ventilatory waves became prominent, as a result of the increase in variations in intrathoracic pressure during artificial ventilation (Sara and Shanks, 1978).

Throughout surgery vasoconstriction—of shorter or longer duration—was evident in the finger in around 75% of our recordings. This was always accompanied by a small increase in systolic arterial pressure. The ear plethysmogram remained stable (fig. 2). These reactions are usually caused by adrenergic responses to stressful stimuli (Goddard, 1982). However, more prolonged vasoconstriction in the finger vascular bed may be part of a general compensation mechanism to an imminent disturbance of the circulation (Dorlas, 1974).

Towards the end of surgery a gradual decrease in amplitude occurred in the finger plethysmogram only (in 80% of our recordings) (fig. 3). This may be attributed to an increase in sympathetic tone during a lighter plane of anaesthesia.

From these observations it is evident that changes in amplitude in the responsive finger plethysmograms are determined by variations in distensibility of the cutaneous vessels under the influence of sympathetic stimuli. However, the reactivity of the vascular bed in the finger can be inhibited by several
anaesthetic agents, as is shown for dehydrobenzperidol in figure 4. Similar effects have been seen with halothane, fentanyl and pethidine (Beddard, 1956; Otteni, Sauvage and Gauthier-Lafaye, 1970; Johnstone, 1974a). The blocking effect of these agents is dose-dependent and varies from patient to patient. Therefore, we feel that the amplitude pattern of the finger plethysmogram may also be used to monitor the effect of “stress-free” anaesthesia. If the blockade is effective, the influence of changes in wall distensibility is eliminated and the amplitude of the plethysmogram will respond to changes in pulse pressure only (Beddard, 1965).

RESPONSES IN EAR PLETHYSMOGRAM

Plethysmograms taken from the pinna were similar to those recorded from the blocked vascular bed in the finger since, in the ear, vasoconstrictor responses are much less pronounced, and only occurred in about 5% of our records. Therefore, the amplitude of the ear plethysmogram will respond mainly to changes in pulse pressure. Figure 5 shows a gradual decrease in amplitude, accompanied by a decrease in systolic arterial pressure, whereas amplitude in the finger and the expired carbon dioxide concentration remained stable. Sometimes, this occurs directly after the beginning of laparotomy and the insertion of the intra-abdominal packs. Since these may interfere with venous return and decrease stroke volume, it suggests a relationship between amplitude in the ear plethysmogram and pulse pressure (Beddard, 1965). However, in a retrospective study of 100 recordings, a significant correlation between amplitude in the ear plethysmogram and pulse pressure could not be substantiated (unpublished observations).

The other factor which determines the plethysmographic amplitude is the distensibility of the vascular walls, but it has been shown already in this article that the vascular smooth muscles in the ear respond minimally to stressful stimuli. Therefore, the distensibility of the vasculature of the ear has to be controlled by mediators other than those in the finger. A difference in control was reported by Johnstone (1974b), who compared the plethysmogram taken from fingers, nose and cheek. Burton (1961) mentioned that there was little evidence of reflex vasomotor control in the skin of head and upper thorax. This “blush area,” however, in contrast to the cutaneous blood vessels of the extremities, demonstrated a predominance of control by circulating pressor agents. Such a control mechanism would agree with the sudden and marked increase in “ear” amplitude we have observed during the final stages of anaesthesia in 42 of 100 patients in whom pethidine was the main analgesic (fig. 6).

The increase in amplitude in the ear plethysmogram occurred spontaneously or after a mechanical stimulus, but without a corresponding further increase in pulse pressure, and there was no correlation with end-expired carbon dioxide concentration. On the other hand, it did seem to be connected with awakening from anaesthesia, and with the drugs used during anaesthesia. Therefore, it is tempting to suggest a relationship with the release of vasoactive agents like catecholamines or central neurotransmitters. These might induce a marked increase in vascular distensibility which only occurs in the ear.
Fig. 6. Example of a sudden and marked increase in ear amplitude in comparison with other variables as the patient regains consciousness.

In conclusion it may be said that changes in the amplitude of the finger plethysmogram may be interpreted as the responses of the peripheral vascular bed to sympathetic stimuli. If this reactivity is blocked, the amplitude of the finger plethysmogram will reflect only changes in pulse pressure. The ear plethysmogram responds minimally to stressful stimuli. It is, therefore, more suitable as a monitor of the systemic circulation and, perhaps, even of stroke volume, but interpretation of changes in amplitude may be influenced by circulating vasoactive substances.

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REFERENCES


