Falciall Valsodilaltion - Blushing

Pages with reference to book, From 54 To 55

Fatema Jawad (Sughra Bai Millwala Hospital, Karachi.)

‘Next came the bride, her cheeks glowing with happiness, is a phrase of fiction which can be interpreted physiologically as a transient vasodilatation of the cutaneous blood vessels. Reddening of the face also called a blush or a flush follows emotions or may be encountered in patients with non-insulin dependant diabetes mellitus, the carcinoid syndrome, in menopause, after glutamate ingestion, thermal induced cr after taking alcohol.

As already established vascular smooth muscles are controlled dually by nerves and catecholamines (Burnstock and Iwayama, 1971., Burnstock, 1975). The nerve fibres are both vasodilator and vasoconstrictor with one of them predominating. Vasodifatation may be acnieved either by inhibiting the nerves causing vasoconstriction being the vasoconstrictor control or by stimulating the nerves giving vasodilatation or the vasodilator control.

Cutaneous blood vessels are found in abundance in the face. (Moretti et al., 1959, Ryan, 1973). These along with the blood vessels supplying the forehead, ears and chest have a vasodilator control which thus brings about an intense flushing reaction in these areas. These nerve fibres originate in the brain stem and travel with the Trigeminal Nerve (Gonzalez et al., 1975) to their destination.

Alcohol induced flushing has been attributed to genetic factors (Wolff, 1972). Blood acetaldehyde levels and ethanol metabolism vary in different races. (Korsten et al., 1975). Acetaldehyde levels explain the enhanced sensitivity to alcohol in Mongoloid patients which is manifested as flushing. It has also been suggested that the intoxication reaction after alcohol in the Japanese may be due to a delayed oxidation of acetaldehyde. (Harada et al., 1980). Flushing reactions seen after ingesting disulfiram in people taking alcoholic beverages has been attributed to the blocking of the alcohol metabolism at the acetaldehyde stage which in turn inhibits dopamine B-hydroxylase and causes vasodilatation. Similar reactions are seen in individuals taking alcohol along with chiorpropamide although no excessive accumulation of acetaldehyde was found. (Fitzgerald et al., 1962). Controversial theories have been put forward regarding the mechanism of this reaction. An increased sensitivity to endogenous opiates has been proposed to be a probable cause (Leslie et al., 1979). The site of action of these opiates is thought to be central (Jeffreys et al., 1979). Another group of workers suggested a prostalandin action probably central in location to give the chiorpropamide alcohol flushing reaction (Horrobin and Manku, 1980).

Flushing is also associated with the carcinoid syndrome. Alcohol has been known to induce flushing in these patients by releasing a catecholamine which acts on the tumour cells which in turn produce a vasodilator (Frolich et al., 1978). This reaction is also provoked by gastrin especially in carcinoids located in the small gut and stomach. Histamine has also been found to be a mediator of gastrin activity. (Black et al., 1972, Gerkens, et al., 1977).

Flushing reactions may be an early evidence of certain other neoplasms too. Basophilic chronic granulocytic leukaemia and systemic mastocytosis give an increased release of histamine which in turn causes flushing. Also excessive production of prostaglandins in systemic mastocytosis and pancreatic tumours as well as carcinoma of the thyroid and renal cell carcinoma leads to flushing. The mechanism is postulated to be a direct action on the vascular smooth muscle by influencing the reactivity to vasoactive substances. (Nasjletti and Malik, 1979, Malik, 1978).

The vasomoter reactions of menopause are well known. The exact mechanism causing it is not yet determined. The decrease in the oestrogen levels and a consequent increase of the gonadotrophins found in menopause could not be proved to be responsible (Yen, 1977, Mulley and Mitchel, 1976, Bullock et al., 1975). The catecholaminergic system dysfunction could be the probable cause of

menopausal flushing. Norepinephrine synthesis increases with a decrease in oestrogen levels. Also norepinephrine influences the production of the leutinizinghormone-releasing factor from the hypothalamus (Simpkins and Kalra, 1979). This factor causes the secretion of the pulsatile leutinizing hormone trom the pituitary which alongwith the affected central thermal regulatory function by norepinephrine (Cox and Lomax, 1977) is responsible for flushing.

Flushing is also seen in some individuals after the ingestion of monosodium glutamate in large doses. This has also been called the 'Chinese Restaurant Syndrome' (Ghadimi et al., 1971). A transient rise in an acetylcholine like substance in sensitive people probably with an inborn error of metabolism is suggested to be the causative factor (Reif-Lehrer, 1976).

Flushing is often encountered after drinking such hot beverages as tea and coffee. The increased heat in the mouth and its surrounding tissues raises the temperature of the blood draining it which eventually enters the internal jugular vein (Wilkin, 1981). This lies parallel to the common carotid artery and an exchange of countercurrent heat raises the the temperature of the blood flowing in the internal carotid artery to the base of the brain where lies the temperature regulating centre. This very sensitive thermostat reacts to small changes in temperature and gives rise to flushing.

The multiple factors producing flushing reactions elucidate the physiological, pharmacological and pathological processes. A further discovery of the underlying cause which could be prealcoholism, a type of diabetes mellitus or a certain type of neoplasia, makes flushing a separate entity from the legendary bridal blush.

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