NORADRENALINE, ADRENALINE, AND THE HUMAN CIRCULATION

BY

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In recent years there has been considerable interest in the circulatory action of the neurohormones, adrenaline and noradrenaline. The effects of these substances in man have been extensively investigated, and it seems appropriate to take stock of this recent knowledge, particularly because it is now possible to define with reasonable certainty the function of adrenaline and noradrenaline in the normal working of the body.

The humoral concept of transmission of nerve impulses dates from the time of Elliott (1905), who suggested that the sympathetic nerves caused their effects by liberating a chemical transmitter substance at their endings which activated the effector cell. Elliott thought that this transmitter was adrenaline, and, indeed, for a long time no other substance was seriously considered in its place. It is surprising that this should have been so, because Barger and Dale (1910), in their classical paper on the chemical structure and sympathomimetic action of amines, pointed out difficulties in accepting adrenaline as the transmitter of sympathetic nerve impulses. They also stated that the action of certain other amines was more like the action of the sympathetic nerves than was that of adrenaline.

The significance of these observations in the past few years has been fully appreciated, for Euler (1946) demonstrated that extracts of organs contained a substance indistinguishable from one of the other amino bases of the catechol group, noradrenaline. This was the most important single observation in support of the theory first put forward by Bacq (1934) that noradrenaline might be the transmitter substance in sympathetic nerves. The immediate task is to describe the action of the two naturally occurring sympathomimetic amines—adrenaline and noradrenaline—in the circulation in man and to define their part in normal and abnormal physiology. Comprehensive reviews covering the chemical and biological action of noradrenaline and adrenaline have recently been published by Euler (1950, 1951) and Goldenberg (1951).

Effects of Adrenaline and Noradrenaline on the Central Haemodynamics

Adequate studies are available to give information on the major changes in haemodynamics caused by adrenaline and noradrenaline. It is probable that the responses obtained during continuous intravascular infusion are of greater value than those obtained after subcutaneous or intramuscular injection, because of the variable absorption time in the latter instance. Therefore, most of the results to be described below are those obtained during infusion. I shall first consider the changes in cardiac output and then proceed to describe the effects of the amines on arterial pressures, overall resistance, and venous pressure. These factors are all interrelated and are altered by the actions of the amines.

The results of all studies agree that, in general, the output of the heart is increased by adrenaline and either unaltered or decreased by noradrenaline. By utilizing the cardiac catheterization technique, McMichael and Sharpey-Schafer

(1944) observed a 50% increase in cardiac output over resting values in subjects given 2 to 5 µg. of adrenaline per minute, while Goldenberg *et al.* (1948), who used doses of 0.15 to 0.30 µg. per kg. per minute, found that the cardiac output was approximately doubled. The latter workers also showed that noradrenaline caused an insignificant change in cardiac output. Similar observations were made by Barcroft and Starr (1951) using a ballisto-cardiograph, but these workers found that noradrenaline caused a small decrease in cardiac output. The change in the cardiac output is in part due to changes in the heart rate and in part due to alterations in stroke volume, for McMichael and Sharpey-Schafer have shown that the increase in the output caused by adrenaline may occur without change in the heart rate. It is also clear that the effects of adrenaline on the circulation may last for 10 to 30 minutes after an infusion, whereas the effects of noradrenaline persist for a much shorter period. In all these studies, intravenous doses ranging from 2 to 35 µg. of adrenaline per minute and from 10 to 35 µg. of noradrenaline per minute were used for periods of 3 to 35 minutes.

Agreement has also been reached on the effects of the amines on the heart rate. Adrenaline uniformly causes an increase in heart rate. During a continuous intravenous infusion two phases of change in the heart rate occur. First, there is a short period during which the heart rate increases to 90 to 120 beats a minute for 30 to 45 seconds. Then the heart rate slows to 5 to 30 beats a minute above the resting value and remains at this level for the duration of the infusion. This secondary slowing of the heart rate does not occur if the subject has been atropinized (Allen, 1948). A bradycardia has been observed with infusions of noradrenaline (Goldenberg *et al.*, 1948; Barcroft and Konzett, 1949; Swan, 1949; Barnett *et al.*, 1950). The bradycardia in the case of noradrenaline and the secondary slowing by adrenaline are due to the vagal influences, for they are abolished by atropine. However, the direct effect of the amines on the heart itself must be considered. In the isolated mammalian heart both amines stimulate the power of contraction and the heart rate (Marsh *et al.*, 1948), adrenaline to a greater extent than noradrenaline. But in the intact human being, as described above, the vagal influences modify the responses. With regard to the changes in cardiac output, it is also clear from Goldenberg's results that, in agreement with the observations on the isolated heart, the stroke volume is increased by both amines.

The pressure changes in the pulmonary artery have been measured by Goldenberg *et al.* (1948), who found an increase in mean pulmonary pressure as measured with the cardiac catheter during infusions of adrenaline and noradrenaline. In the systemic circulation, however, the changes caused by these amines differ. Adrenaline causes an increase in systolic pressure, but its action on the diastolic pressure is variable; at times there may be a fall, but there may be either no change or an increase in pressure. In any case, there is an increase in pulse pressure and only a moderate increase in mean pressure. Noradrenaline increases systolic, diastolic, and mean pressures, the pulse pressure remaining unaltered.

From the data on cardiac output and systemic arterial pressure the effective resistance to the ejection of blood from the heart may be calculated from the following equation: $T.P.R. = \frac{P}{C.O.}$; where T.P.R. = total peripheral resistance, C.O. = cardiac output, and P = mean blood pressure. The major variable factor in the total peripheral resistance is the overall state of tonus of the blood vessels. Briefly, adrenaline decreases the T.P.R. while noradrenaline causes an increase in this value (Goldenberg *et al.*, 1948). Therefore one may say that adrenaline in small doses is a dilator substance while noradrenaline causes an overall constriction. Examination of pulse contour tracings obtained from the brachial artery supports these results. Whereas adrenaline causes a sharp fall of the descending limb of the pulse contour and a depression of the diastolic

notch, *noradrenaline* causes a flattening of the descending limb and an elevation of the diastolic notch. The rate of fall of pressure during diastole is a qualitative index of peripheral resistance.

Both *adrenaline* and *noradrenaline* cause a rise in peripheral venous pressure (Iglauer and Altschule, 1940; Fowler *et al.*, 1951). The latter authors have also demonstrated that the pulmonary venous pressure and right auricular pressure are increased during infusions of *noradrenaline*.

Effect of Adrenaline and Noradrenaline on the Local Dynamics

The overall vascular changes have their counterpart in the different organs of the body, for the sum of the blood flow through the organs must equal the cardiac output. Blood-flow studies at rest have been carried out on the more important organs, and the results obtained during intravenous infusion are in keeping with the changes in total peripheral resistance already described. The Table shows the blood-flow studies carried out and the responses obtained during infusion of *adrenaline* and *noradrenaline*.

Effect of Intravenous Administration of Adrenaline and Noradrenaline on the Blood Flow in Various Organs (After H. Barcroft)

Organ	Blood Flow				
	Before Admin- istration of Adrenaline or <i>Noradrenaline</i>	During Administration of Adrenaline		During Administration of <i>Noradrenaline</i>	
	ml./ Minute	ml./ Minute	Change %	ml./ Minute	Change %
Liver ¹	800	1,600	+100	800	0
Kidney ²	1,500	900	-40	1,200	-20
Skeletal muscle ³ ..	1,000	2,000	+100	1,000	0
Brain ⁴	750	900	+20	675	-10
Total flow ..	4,050	5,400	+30	3,675	-10

¹ Bearn, Billing, and Sherlock (1952).

² Barclay, Cooke, and Kenney (1947), and Barnett, Blacket, Depoorter, Sanderson, and Wilson (1950).

³ Duff and Swan (1951).

⁴ King, Sokoloff, and Wechsler (1952).

The effect of the amines on blood flow in the muscles has been investigated extensively because of the relative ease with which such studies can be carried out. In the case of *adrenaline*, the response is complex and will be described in some detail. When *adrenaline* is infused directly into the main artery of a limb and the resultant changes in blood flow are measured, the direct effect of *adrenaline* on the tonus of the blood vessels may be observed, for the pressure in the arterial system will remain constant and little *adrenaline* will reach the general circulation. In these circumstances *adrenaline* causes a dilatation which lasts 45 to 60 seconds (Allen *et al.*, 1946). Then the blood flow returns to about its resting level. Larger doses of *adrenaline* cause constriction of the blood vessels of the muscles after the transient dilatation has passed off. It is worth noting that sufficient amounts of *adrenaline*, given intra-arterially, to constrict the blood vessels of muscles also cause effects in the general circulation (Swan, 1951a). The immediate local dilatation caused by *adrenaline* in muscle is so transient an effect that its physiological significance may be open to doubt.

When *adrenaline* is given intravenously, however, the transient dilatation is followed by a phase of sustained increase in blood flow. When *noradrenaline* is infused into the femoral artery the dose required to produce constriction of the vessels of the muscles is only a tenth to a twentieth as large as the dose of *adrenaline* required to produce an equal degree of constriction when administered in the same manner. Transient vasodilatation is not caused by infusion of *noradrenaline*, nor does sustained dilatation occur when this amine is given by intravenous infusion. It is of interest to note that, whereas *adrenaline* causes an

inhibition of sympathetic vasoconstrictor tone (Swan, 1951b), in the case of *noradrenaline* this inhibition was not demonstrated with similar techniques (Swan, 1951c).

Physiological Role of Noradrenaline and Adrenaline

Noradrenaline now appears to be generally accepted as the predominant transmitter substance liberated at sympathetic nerve endings. Although the greater part of the evidence for this was obtained from animals, it is unlikely that man differs from other mammals in this respect. Euler (1950) obtained *noradrenaline* from the human sympathetic chain, and Holton (1949) and Goldenberg, Faber, *et al.* (1949) have demonstrated large amounts of *noradrenaline* in tumours of the suprarenal medulla. *Noradrenaline* is excreted in small amounts in both the free and the conjugate form in the urine of normal persons. Also, considering the responses occurring in blood vessels of muscles described in the preceding section, it is clear that in man *noradrenaline* is a much more efficient constrictor mediator than *adrenaline*.

The concept of *noradrenaline* as the principal mediator of sympathetic constrictor impulses now replaces the sympathin theory of Cannon and his associates. Euler (1950) states: "... *Noradrenaline* as the chief active principle in the adrenergic nerves has assumed a central position in autonomic nerve physiology. This, however, at the same time conduces to bear out Cannon's theory that *adrenaline* in the first place should be regarded as a specific hormone serving emergency functions." If *adrenaline* is a hormone with a general action, *noradrenaline* may be contrasted as a substance which acts at or near the site of its liberation. Only small amounts of *noradrenaline* leak away from the neurovascular junction, and these cause no significant effect on the general circulation. It is probable that *noradrenaline* is liberated in very small amounts at all sympathetic nerve endings, and thereby maintains the vasomotor tone and the peripheral resistance.

The nerves to the skin and the cardiac sympathetic nerves are cited as possible exceptions to this generalization, although *noradrenaline* acts as a cardiac stimulant and as a coronary vasodilator (Marsh *et al.*, 1948; Burn and Hutcheon, 1949). But the cardiac nerves could also mediate their effect by liberation of *noradrenaline*, for the local liberation of *noradrenaline* in the heart should cause the same stimulatory effects seen in the isolated mammalian heart. The reflex inhibitory effects seen during intravenous infusion of *noradrenaline* in man are caused by the rise in arterial blood pressure due to overall vasoconstriction, an event which would not result from the local liberation of *noradrenaline* at the endings of the cardiac nerves.

Cannon's views (1940) on the emergency function of the action of *adrenaline* still hold good. Indeed, the recent work of investigators on the human subject has done much to indicate the role of *adrenaline* in normal physiology which he first propounded. I have given intravenous infusions of *adrenaline* to more than 60 different persons and have asked them if and how the symptoms they experienced compared with their emotions during times of alarm in the past. In the majority of cases the symptoms were similar, although usually they exceeded the stressful sensations in severity. I would judge from the replies that *adrenaline* enters the circulation during stress, but at rates rather below 10 µg. a minute. In contrast to *adrenaline*, the milder symptoms caused by *noradrenaline* were always unfamiliar to the subjects, although fear occasionally does occur when this substance is administered.

The secretion of the suprarenal glands is a mixture of both *adrenaline* and *noradrenaline*, the content of *noradrenaline* being up to 25% of the total. But this mixture will fundamentally have the action of *adrenaline*, for de Lary *et al.* (1950) have shown that, even with equal amounts of the amines, the effects of *adrenaline* predominate. *Adrenaline* then is liberated from the suprarenal

glands either paroxysmally or over short periods of time during stressful circumstances, and causes an increased cardiac output and a redistribution of blood flow. Adrenaline has a powerful excitatory action on the heart, for it maintains an increase in heart rate in spite of vagal action.

Role of Noradrenaline and Adrenaline in Disease Processes

Because noradrenaline is now regarded as the principal transmitter substance of sympathetic nerves, its possible role as the causative agent in human hypertension is obvious. Goldenberg *et al.* (1948) suggested that essential hypertension might be due to a failure of methylation of noradrenaline to adrenaline, and thus an excess of the constrictor hormone, noradrenaline, would accumulate at the sympathetic nerve endings. Other possibilities exist. In a small series of cases of hypertension recently investigated the blood vessels of muscles appeared to be more sensitive to the constrictor action of noradrenaline than did the vessels of normal persons. The interpretation of the significance of this was complicated by the large variation that occurs in normal persons, and the observation serves only to indicate the possibility that a sensitivity to noradrenaline may exist in hypertensive vessels. Goldenberg *et al.* (1948) observed that intravenous administration of noradrenaline caused a greater rise in systemic blood pressure in patients who had hypertension, and this was also found by Barcroft, Konzett, and Swan (1951), although Judson, Epstein, and Wilkins (1950) reported that the percentage change in blood pressure resulting from intravenous injection of noradrenaline was the same in patients who had hypertension as it was in normal persons. Intravenous injection of adrenaline causes much less change in blood pressure in hypertension, and the transient dilatation in muscle is, paradoxically, less evident than in normal persons, although in some patients the dilator response is clearly seen (Barcroft *et al.*, 1951). It would be unwise to go further than to suggest that the vessels in cases of hypertension may react in an abnormal way to the naturally occurring sympathetic neurohormones and that, if this is shown to be a consistent finding, it may be of importance in the aetiology of human hypertension.

In regard to the vasospastic diseases, there is at present no conclusive evidence that the neurohormones are concerned with the cause of these conditions, although at one time this appeared likely. In normal subjects the response of the cutaneous blood flow varies widely at different initial levels of flow, and a careful comparison of the direct constrictor effects of adrenaline and noradrenaline in normal persons and patients with vasospastic disease is required to evaluate this situation.

In pheochromocytoma of the suprarenal gland a definite relationship with the sympathetic neurohormones has been demonstrated. In this condition a tumour of chromaffin tissue commonly exists either in the suprarenal gland itself or in the para-aortic region. The tumour is usually a benign adenoma, but in about 10% of cases it is malignant.

When this tumour is present the physiology of the suprarenal gland differs from normal in the following respects. First, the total amount of pressor hormone secreted in response to stimulus is far in excess of normal. Also, a high proportion of noradrenaline may be produced, so that the effect of the mixture of amines is that of noradrenaline and not that of adrenaline. There is evidence, too, that in many cases the mixture of hormones is liberated from the suprarenal gland continuously over long periods of time, in contrast to the transient response to stress.

The excessive amounts of adrenaline liberated give rise to a qualitatively different circulatory reaction from that described in an earlier section concerning the action of physiological amounts of adrenaline. The response in cases of pheochromocytoma has been well compared to the "pharmacological" effect of adrenaline with marked hypertension (Goldenberg, 1951). It cannot be too strongly emphasized that in the normal subject adrenaline as a

physiological substance is not a hypertensive agent. Only in these abnormal states with large amounts of circulating adrenaline is there an overall vasoconstriction with increased total peripheral resistance, and raised diastolic, systolic, and mean blood pressures. If the amount of adrenaline is grossly excessive and the patient survives the acute hypertensive state, then a state of peripheral vascular collapse similar to that observed in dogs (Viggran and Essex, 1950) may supervene. Frequently, the removal of a pheochromocytoma is followed by an acute hypotension due to the sudden removal of the constrictor hormone, which by its presence in the circulation has maintained the overall vascular tonus. In these respects the action of adrenaline is modified in cases of pheochromocytoma.

Noradrenaline is not normally an effective circulating agent, for its action is localized to the nerve ending, but when elaborated by medullary tumours and liberated into the circulation it causes effects similar to those seen during its infusion in man. In excess it causes a moderate increase in metabolism with raised blood sugar and raised metabolic rate. Goldenberg, Aranow, Smith, and Faber (1950) pointed out that small tumours with a high noradrenaline content mimic essential hypertension, but that larger tumours, in which adrenaline is also predominant, cause hypermetabolic symptoms in addition. Finally, Calkins *et al.* (1950) suggested that excessive amounts of hormone initiate a mechanism whereby a more permanent hypertension is caused, no longer due directly to circulating adrenaline or noradrenaline. Also, it is possible that permanent damage to certain organs may be responsible for the persistent hypertension which is found in some patients after removal of a pheochromocytoma.

Adrenaline and Noradrenaline as Therapeutic Agents

For many years adrenaline has enjoyed a prominent place in the therapy of hypotension. Because of the selective vasoconstriction in the vessels of the skin, the rise in blood pressure which follows intramuscular injection of adrenaline was thought to result from vasoconstriction. This is not the case, for, as Allen (1948) has shown, doses of 0.5 mg. of adrenaline given intramuscularly considerably increase the blood flow in the forearm, and the results of Starr *et al.* (1937) indicate that the changes in the cardiac output and blood pressure are similar to those obtained during continuous infusion—that is, consistent with a diminished peripheral resistance.

The rise in blood pressure is due to an increased cardiac output and possibly to a venoconstriction augmenting the venous return to the heart. In cases in which vasomotor collapse is due to an arteriolar dilatation, therefore, adrenaline would not seem to be the agent of choice, for it would do little to restore the peripheral resistance. But its undoubted resuscitative effect in such cases is probably due to its effect on the venous return to the heart as well as to cardiac stimulation. On physiological grounds, noradrenaline would appear to be the agent of choice in peripheral circulatory collapse, for it causes an overall vasoconstriction and increases the total peripheral resistance. Goldenberg, Apgar *et al.* (1949) first published results obtained by using noradrenaline as a pressor drug in a series of patients undergoing thoraco-lumbar sympathectomy for hypertension. Intravenous infusion of noradrenaline overcame the acute hypotensive state which frequently occurred during and after this operation. Since then, noradrenaline has been often administered in doses of 5 to 40 μ g. a minute in the treatment of many types of peripheral vasomotor collapse, including that which follows removal of pheochromocytoma (Swan, 1951d; Cahill and Monteith, 1951), and in the treatment of acute hypotension following ganglionic blockade with pentamethonium and hexamethonium (Churchill-Davidson, 1951). In general, the response has been satisfactory, but it remains to be shown whether or not noradrenaline possesses many practical advantages over the more usual pressor drugs. The use of noradrenaline may require more attention than other

pressor drugs—for example, desoxyephedrine—but such care is, surely, the first requirement of any patient in an acute hypotensive state.

Summary

Noradrenaline, the transmitter substance of the sympathetic nerves, has a predominantly local action and its steady release at the nerve endings causes constriction of the local blood vessels. The overall activity of the sympathetic nerves results in the normal peripheral vasomotor resistance, which is a major factor in the maintenance of the blood pressure.

Adrenaline is an "emergency substance" liberated into the circulation and acting generally. Under physiological conditions it causes an overall vasodilatation and an increase in cardiac output.

Only in phaeochromocytoma of the suprarenal gland has a direct relationship with disease been established with the neurohormones, adrenaline and noradrenaline.

Noradrenaline would appear to be the most rational agent in the treatment of the hypotensive state due to peripheral collapse.

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Addressing the Institute of Almoners at B.M.A. House on March 28, Miss M. P. Hornsby-Smith, Parliamentary Secretary to the Ministry of Health, said that it had become increasingly clear that the hospital almoner was now one of the most important contributors to rehabilitation. The work of the physician or surgeon and of the authorities responsible for ensuring that people who had been ill were restored to health and able to find and keep jobs would be seriously hampered were it not for the presence of the almoner. In the National Health Service they regarded rehabilitation as one of the most promising developments of the present time—not least because it enabled them to make a more effective use of the hospital beds and out-patient facilities available.

STATURES OF A GROUP OF UNIVERSITY STUDENTS AND OF THEIR PARENTS

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Although there is an abundance of statistical material relating to the statures of populations of widely differing ages, there have been remarkably few attempts to compare directly the heights of a group of parents and of their offspring. Two exceptions, however, are reported in the papers by Pearson and Lee (1903), and by Bowles (1932).

There is no doubt that present-day children are growing faster than those of previous generations. But is the final adult stature to-day greater than the adult stature in the past? Authoritative British opinion at the moment is against the view that adults to-day are taller than adults in the past. However, Pearson and Lee (1903) in this country, and Bowles (1932) in the United States of America, came to the conclusion that there was some increase in adult stature in the second generation.

At a Conference Meeting of the Nutrition Society in 1950 Dr. B. Woolf suggested that in view of these contradictory conclusions another survey of the heights of young adults and of their parents would be interesting (Weir, 1951).

Accordingly it was decided to compare the heights of the students attending the course in physiology at Glasgow University with those of their parents.

Methods

The students were measured in the Institute by one of us (J.D.), using a wooden calliper. Then in groups of two to four the students were given explicit detailed instructions and a demonstration of how to measure their parents. They took home with them special 7-foot (213-cm.) inextensible inch tapes and a printed card containing a list of the information required.

There were 330 students in the physiology classes. In 38 instances one or both parents were not alive, and another 45 students were living away from home and thus unable to see their parents for some months. The remaining 247 undergraduates, 63 women and 184 men, all domiciled in Glasgow or within a radius of about 30 miles (48 km.), were included in the investigation.

Results

The results showed that 76.2% of the women students were taller than their mothers and 73.9% of the men taller than their fathers. The differences of these percentages from the expected 50% on a null hypothesis—that is, that there has been no increase in stature—are very highly significant ($P < 0.001$). Even if we restrict ourselves to comparing the number of students who are an inch (2.5 cm.) or more taller than their parents with those who are less than an inch (2.5 cm.) taller, the result is still highly significant ($P < 0.01$).

The mean height of the women students was 64.4 in. (163.6 cm.)—S.D. 2.0 in. (5 cm.)—and of their mothers 62.7 in. (159.3 cm.)—S.D. 2.1 in. (5.3 cm.)—a difference of 1.7 in. (4.3 cm.). The 95% fiducial limits of this difference are 2.44 and 0.96 in. (6.2 and 2.4 cm.).