Pressor and Depressor Responses to Tilting in Hypertensive Patients

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The responses of intra-arterial blood pressure to passive head-up and head-down tilting are described for 50 patients with well established hypertension and compared to results obtained in subjects with normal cardiovascular systems. This study indicates that abnormal depressor and pressor responses are present in many patients with hypertension, that depressor reflexes are independent of pressor reflexes, that marked variations of depressor and of pressor reflex activity occur between hypertensive patients, but that the combination of depressor-pressor responses in a given patient usually remains constant.

In 1895 Leonard Hill concluded from his experimental work on the blood pressure and heart rate of animals that the effect of changing the position afforded a most delicate test of the vasomotor mechanism. The development of accurate methods of continuous recording made it possible to confirm these conclusions in man.

Previous studies of normal subjects established a definite pattern of blood pressure and heart rate in response to tilting. During a tilt, an immediate hydrostatic change of blood pressure always occurred at the level of the aortic arch due to the effect of gravity on the central blood column. Thus, during the tilt to the head-down position the blood pressure rose and during the tilt to head-up position the blood pressure fell. The elevated pressure produced by the head-down tilt was followed by a depressor response and the fall of pressure produced by the head-up tilt by a pressor response. In both instances the blood pressure returned to approximately the same level for an individual within 18 sec. regardless of position. Comparable results to head-down tilting have been reported by Wilkins, Bradley, and Friedland.

The present report deals with similar studies in 50 patients with hypertension. The hydrostatic alterations of pressure during tilting were similar to those obtained in normal subjects. However, following tilting, striking and varied differences from the normal reflex pattern often occurred.

Method

Intra-arterial radial blood pressure was recorded continuously during and following tilting in 50 ambulatory patients with sustained hypertension and compared with the records obtained previously in 20 normal subjects.

Forty-five patients were tilted at a moderate rate (5 sec. per tilt) between the 30° head-up and the 30° head-down positions. Each position was maintained for at least 20 sec. Five patients in earlier experiments were tilted between the 20° head-up and the 45° head-down positions. The tilt to each position was repeated at least 3 times in all patients. In addition, the blood pressure was usually recorded in the horizontal position before and following tilting. Ten of these patients were restudied on one or more occasions after periods of time that varied from 1 week to 18 months.

The degree and rate of tilting remained constant by means of an electrically operated table. Braces supported the patients securely, so that they were comfortable throughout the tilt and no muscular activity was required to maintain the position. The arm was placed on a support in the plane of the table top perpendicular to the body whereby the radial artery remained at the level of the shoulder throughout the tilt. Pressures recorded with the arm in this position indicated pressure changes at the level of the aortic arch.

The blood pressure was determined in the radial
artery by means of a Statham strain gage adapted to record through a string galvanometer electrocardiograph. A 1½-inch, 20-gage needle was used for the arterial puncture.

Results

Normal Subjects. Hydrostatic changes in blood pressure occurred during tilting (see fig. 1 and 2). With the arm maintained at the shoulder level the blood pressure always rose during movement to the head-down position (average rise 19/16 mm. Hg) and fell during the movement to the head-up position (average fall 14/13 mm. Hg).

A depressor response followed the hydrostatic elevation of head-down tilt (average fall 13/13 mm. Hg); a pressor response followed the hydrostatic fall of the head-up tilt (average rise 13/13 mm. Hg). In both positions these responses returned the blood pressure within 18 sec. to a level differing not more than 5/5 mm. Hg from the initial pressure. During the depressor response the blood pressure did not fall more than 5/5 mm. Hg lower than the initial head-up pressure. During the pressor response the blood pressure did not rise more than 5/5 mm. Hg above the initial level. Each subject showed similar response on successive tilts. Five subjects studied at intervals of 1 to 3 weeks had no change in response to tilting.

Hypertensive Patients. Hydrostatic changes in blood pressure always occurred during tilting (see fig. 1 and 2). The average elevation during the head-down tilt was 19/17 mm. Hg. The average fall during the head-up tilt was 19/15 mm. Hg.

In 11 patients the depressor-pressor responses following tilting conformed to the normal pattern. Five patients were more than 70 years old; of these 3 had known hypertension for at least 10 years. The other 6 patients had sustained no apparent damage to the brain, kidneys, or heart.

There were 7 additional patients who had a normal depressor response. In 5 of these, however, the pressor response was increased and
in the other 2 the pressor response was decreased.

In 4 patients almost no depressor or pressor response occurred, i.e., the blood pressure following the tilt remained at the level produced by the hydrostatic effect of the tilt. One of these patients had a pheochromocytoma. A second patient died of uremia and at autopsy had arteriolar nephrosclerosis. A third patient had severe congestive heart failure complicated by renal insufficiency. In the fourth patient, hypertension had developed during pregnancy.

There were 22 patients in whom a decreased depressor response occurred following tilting to the head-down position, i.e., the blood pressure after 18 sec. in the head-down position was more than 5/5 mm Hg above the blood pressure recorded in the erect position. In these patients pressor activity following the head-up tilt varied. It was classified as normal in 7, increased in 4, decreased in 8, and absent in 3. In this group were 6 patients who sustained 1 or more cerebrovascular accidents and 4 who had severe retinal hemorrhages.

An increase in depressor activity was found in 6 patients. In this group the blood pressure rapidly fell within a few seconds following the head-down tilt to a level that was more than 5/5 mm Hg lower than in the head-up position. The reading in 1 patient was 33/11 mm Hg lower than the erect pressure 9 sec. after the head-down tilt. Patients with this type of response invariably complained of dizziness with bending down and similar maneuvers.

Pressor response following tilting to the head-up position was absent in 8 patients and in 13 patients it was decreased, in that the pressure 18 sec. after the tilt remained more than 5/5 mm Hg lower than the initial blood pressure. Dizziness on standing was a common complaint in the patients who had diminished or absent pressor responses following tilting to the head-up position. It was not observed in the other patients.

In 10 cases the pressor response was increased in that blood pressure shortly after the head-up tilt rose rapidly to a level of more than 5/5 mm Hg above the initial erect pressure.

Blood pressure readings were recorded during a complete cycle of tilting at least 3 times during these tests. In addition, there were 10 patients who were tested at intervals from 1 week to 18 months. The combination of depressor-pressor responses observed in a patient remained the same both on successive tilts and over an interval of time, except in 1 patient in whom a bilateral dorso-lumbar sympathectomy, D-11 to L-2, was performed. Preoperatively, this patient had a pressure of 310/173 mm. Hg. Her depressor response was normal; the pressor response was increased. One year after the operation the blood pressure was 215/130 mm. Hg. Her depressor response remained active; the pressor response was markedly diminished.

The independence of depressor and of pressor activity is demonstrated by the lack of correlation between the type of pressor and the type of depressor response in these patients. There were 18 patients with a normal depressor response; the pressor response was normal in 11 of these, decreased in 2, and increased in 5. There were 22 patients with a decreased depressor response; the pressor response was absent in 3 of these, normal in 11, decreased in 8, and increased in 4.

**Discussion**

Criteria chosen to classify a response as normal or abnormal were selected after analysis of the control records of normal subjects. Whereas other points of comparison might have been used, the constancy of the response at 9 and 18 sec. in the control subjects suggested comparison with similar readings obtained in hypertensive patients.

The division of response into types as used in the presentation of data is admittedly somewhat arbitrary. There were some hypertensive patients with a response that conformed to the control pattern and some in whom the separation of normal from abnormal depended on a difference of 1 to 2 mm. Hg. In the majority of the patients, however, the differences from the control responses were striking and they remained constant on repeated studies.

Apparently the depressor and pressor responses were at least partially independent of each other, since there was often variation between the type of depressor and the type of
pressor activity in a patient, e.g., one might find decreased depressor response with either absent, decreased, normal, or increased pressor response. If a single mechanism produced these adjustments, a positive correlation between the types of response would be expected. The concept of independence of depressor and pressor response has been supported by finding that dorsolumbar sympathectomy abolished the pressor activity following tilting, whereas the depressor response remained active. We have also found that atropine diminished depressor activity in normal subjects without interfering with the pressor response. These changes after sympathectomy and atropine indicated that the depressor-pressor responses were mediated by a reflex rather than a humoral mechanism. In addition, the onset of adjustment in control subjects occurred within a few seconds of the start of the tilt. It is hard to visualize a humoral mechanism efficient enough to reverse itself repeatedly as abruptly as occurred in the control subjects.

Several hypotheses were suggested by the observations:

1. Normal or overactive responses were usually obtained in patients who were younger or whose hypertension had caused little obvious damage, while diminution of reflex activity was associated with later, more serious manifestations of the disease such as cerebrovascular accident and retinal hemorrhage. This suggests that changes in response were associated with a complication of hypertension that in turn contributed to the seriousness of the disease.

2. Heymans and associates produced hypertension in animals by interfering with the depressor mechanism. Procaine block or section of the carotid sinus nerve in human beings resulted in temporary elevations of blood pressure. In our series, the depressor response was diminished or absent in 26 patients. The hypertension in some of these cases may have been related to hypofunction of the "buffer mechanism."

3. Finally, it is well established that sympathectomy in man may lower blood pressure. Ten patients had an increased pressor response; this may represent an overactive sympathetic nervous system that may have been an etiologic factor in their hypertension.

Our results confirm the well-known fact that dizziness on standing may be associated with diminished pressor activity. In addition, an increased depressor response may explain the paradox of dizziness while in a bent-over position.

The pattern of depressor-pressor activity was not related to the level of the blood pressure. For instance, normal responses occurred in 1 patient who had a horizontal blood pressure of 317/175 mm. Hg, whereas the reflex responses were absent in another patient whose horizontal pressure was 180/120 mm. Hg.

**SUMMARY**

Responses of blood pressure to tilting have been studied in 50 hypertensive patients and compared with findings in 20 normal subjects.

A normal depressor and pressor response occurred together in 11 patients. Variations from the normal pattern were noted in 39 patients in whom either or both responses were increased, decreased, or absent. Absent or diminished depressor activity was present in 26 patients.

Independence of depressor and pressor response has been demonstrated. Evidence has been presented that depressor and pressor responses are mediated by a neural mechanism.

The data suggest that disorder of reflex depressor as well as of pressor mechanism may be important in the etiology of hypertension.

**SUMMARIO IN INTERLINGUA**

Responsas al position in plano inclinare esseva studiate in le pression sanguinee de 50 patientes hypertensive. Le datos esseva comparate con constatationes correspondente in 20 subjectos normal.

Le combination de normal responsas presorial e depressorial occureva in 11 patientes. Deviationes ab le norma esseva notate in 39 patientes. In illes le un o le altere responsa o ambe responsas esseva augmentate, reduce, o absente. In 26 patientes le activitate depressorial esseva reduce o absente.

Esseva demonstrate le independentia del responsas depressorial e presorial. Es presentate observationes in supporto del conception
que le responsas depressorial e pressorial es mediate per un mecanismo neural.

Le datos presentate pare indicar que disor-dines del mecanismo del reflexos depressorial e pressorial pote esser importante pro le etiologia de hypertension.

REFERENCES

1 Hill, L.: The influence of the force of gravity on the circulation of the blood. J. Physiol. 18: 15, 1895.


Perera, G. A.: Edema and Congestive Failure Related to Administration of Rauwolfia serpentina.


In 5 patients with hypertensive vascular disease, fluid retention occurred after the administration of crude or pure alkaloids of Rauwolfia serpentina. None of these patients had been in failure prior to the administration of rauwolfia and all cleared when treatment ceased. One of the patients who had developed marked edema while receiving reserpine was again given the drug for a week’s time. Definite pitting edema of the ankles was again apparent. It vanished once more when administration of reserpine was discontinued. In 2 patients fluid retention was severe enough to cause congestive failure. The occurrence of sodium retention during therapy with rauwolfia der- ivatives should be kept in mind whenever edema or congestive failure develops in patients who are taking such medications.

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