The drug had a marked effect in reducing gastric motility and tone.

There was a smaller effect in reducing acidity of the gastric contents.

Tricyclamol prolonged the reduction of gastric acidity obtained by a dose of alkali.

We wish to thank Dr. J. D. Dow for the *x*-ray films, Professor W. R. Spurrell for providing facilities for the analysis of test meals, and Messrs. Burroughs Wellcome and Co. for supplies of the tricyclamol chloride used in these investigations.

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EFFECTS OF VALSALVA'S MANŒUVRE ON THE NORMAL AND FAILING CIRCULATION

BY

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The Valsalva manœuvre (Valsalva, 1707), by producing a rise in intrathoracic pressure, causes an acute decrease in the effective filling pressure of the heart, with consequent changes in the stroke output. An analysis of continuous arterial pressure tracings in the normal subject performing the Valsalva manœuvre has shown that a decrease in pulse pressure occurs when intrathoracic pressure is raised, and that this is followed by a phase of raised diastolic pressure (the "overshoot"). The latter phase has been interpreted as a baroceptor response to the decreased pulse pressure of the former (Sharpey-Schafer, 1953).

Cases of heart failure might be expected to respond to the Valsalva manœuvre in a different manner, since a decrease in effective filling pressure produced by venesection may cause an increase in stroke output (Howarth *et al.*, 1946). Preliminary observations, using continuous arterial pressure records, indicated that this was in fact so (Sharpey-Schafer, 1952).

This paper presents the accumulated data on normal subjects and observations on cases of heart failure of varied aetiology. The results have been interpreted in terms of baroceptor responses to acute changes in pulse pressure.

Material and Methods

Sixty-two subjects with a normal circulation and 63 cases of heart failure were studied. All the cases of heart failure were in sinus rhythm and had a jugular venous pressure raised from 2 to 25 cm. above sternal angle level: 19 were cases of hypertensive heart failure, 9 had ischaemic heart disease, 21 had disease of the mitral or aortic valves, 7 had cor pulmonale (emphysema), 3 severe anaemia, 2 severe thyrotoxicosis, and there was 1 each of the following: primary pulmonary hypertension, pulmonary embolus, haemochromatosis, and chronic myocarditis.

Continuous arterial pressure records were obtained using capacitance manometers (Hansen, 1949), which were also employed for the measurement of intrathoracic pressure (Dornhorst and Leathart, 1952). In some observations a differential manometer system was used (Lee *et al.*, 1954), usually differentiating intra-arterial and intrathoracic pressures.

The Valsalva manœuvre was performed by blowing a mercury column to 40 mm. Hg and maintaining it at that level for about 10 seconds. This was easily accomplished by patients with heart failure, however breathless, and by quite young children for a small monetary reward. The procedure is without risk in heart failure for reasons that will become apparent. All subjects were supine with the trunk raised to 45 degrees with the horizontal.

Results

The difference in response between normal subjects and cases of heart failure was conspicuous (Figs. 1, 2, and 3).

Factors Affecting Response in the Normal Subject

When the intrathoracic pressure is suddenly raised to 40 mm. Hg this pressure is rapidly transmitted to all the arteries and to those veins without valves. The immediate supply pressure to many internal organs, such as the brain, is therefore essentially unaltered. In skin and muscle, however, the venous valves shut and initially the arteriovenous supply pressure is raised by approximately the same amount as the intrathoracic pressure. Since the blood-flow to a part depends on supply pressure and the calibre of the arterioles,

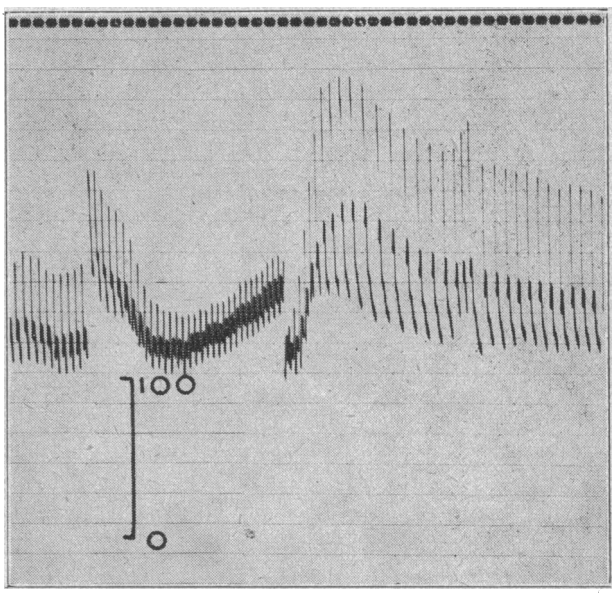

FIG. 1.—Continuous arterial record showing effect of Valsalva manœuvre in normal subject. Calibration in mm. Hg and time marker in seconds in this and subsequent records.

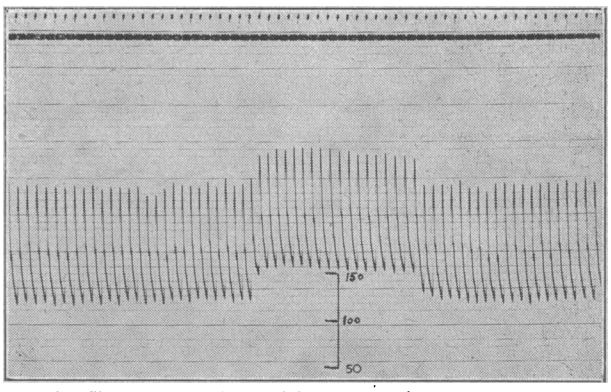

FIG. 2.—Hypertensive heart failure, showing effect of Valsalva manœuvre on arterial blood pressure.

the blood-flow to skin and muscle is initially increased. Thereafter the local venous pressure rises steadily at a rate which depends on previous vasomotor tone, the rise being more rapid in a hot limb than in a cold limb (Fig. 4); as the arteriovenous pressure difference falls the blood-flow to the part decreases. These effects may be seen directly in the capillaries of the nail-bed.

It has already been observed that the accumulation of blood in peripheral veins behind closed valves causes the central effective venous pressure to fall (Lee *et al.*, 1954), with a consequent decrease in stroke output and a fall in arterial mean blood pressure and pulse pressure. The rate at which mean arterial pressure and pulse pressure will decrease depends, therefore, on the initial state of vasomotor tone, being more rapid and considerable in the vasodilated subject. It was shown previously that the rate of fall increased after hyperventilation, which causes vasodilatation of muscle vessels (Howard *et al.*, 1951). The same results have since been found in vasodilatory states produced by indirect heating and by sympathetic blocking agents, and in cases of thyrotoxicosis. It has also been shown that if the rate of accumulation of venous blood in the periphery is decreased by cold or by a pressure suit, then arterial mean pressure and pulse-pressure changes are also decreased.

Under normal environmental conditions the arterial pressure begins to rise again after 5 to 10 seconds, with the onset of peripheral constriction (Sharpey-Schafer, 1953). Simultaneously, there is a decrease or stoppage of blood-flow in the capillaries of the nail-bed.

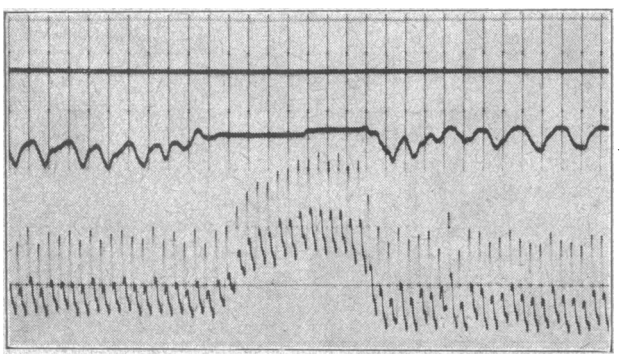

FIG. 3.—Severe hypertensive heart failure, showing increase of pulse pressure during Valsalva manœuvre and decrease of diastolic pressure after release of the raised intrathoracic pressure.

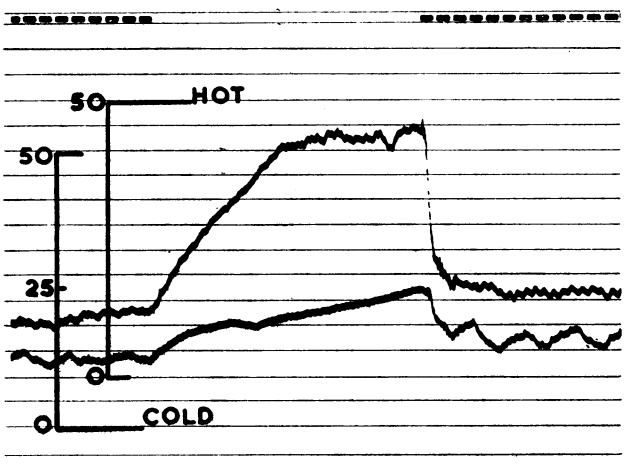

FIG. 4.—Simultaneous venous pressure records from the antecubital veins of both arms of a normal subject. The upper curve is from an arm previously heated; the lower from an arm at a cool environmental temperature. The Valsalva manœuvre is signalled by an interruption of the time marker. The venous pressure rises more rapidly in the heated limb.

The results are summarized in the "normal" part of Fig. 5, in which maximum percentage change in arterial pulse pressure during the phase of raised intrathoracic pressure is plotted against the maximum change in diastolic pressure during the "overshoot." There is a direct relationship in

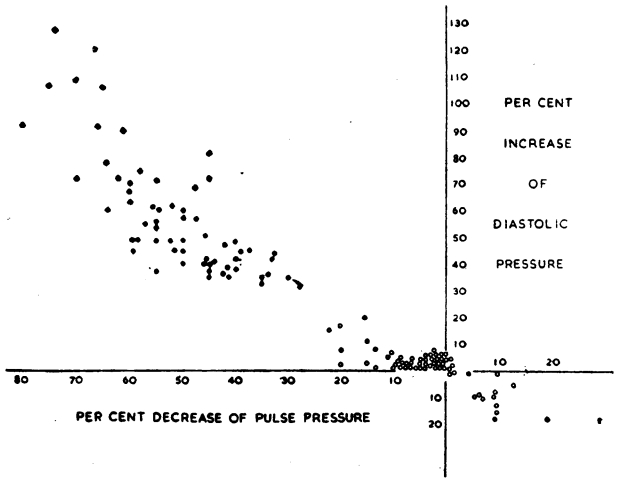

FIG. 5.—Normal subjects—black circles; cases of heart failure—open circles.

$$\text{Percentage decrease of pulse pressure} = \frac{\text{Control pulse pressure} - \text{maximum change in pulse pressure during Valsalva}}{\text{Control pulse pressure}} \times 100$$

$$\text{Percentage increase of diastolic pressure} = \frac{\text{Maximum change in diastolic pressure after Valsalva} - \text{control diastolic pressure}}{\text{Control diastolic pressure}} \times 100$$

the normal subject. In each individual a series of points may be obtained on the "normal" part of the curve by altering the initial vasomotor tone by methods described above.

Response in Heart Failure

The results fall into two groups. In the first, comprising the majority of the cases, there was no measurable change in pulse pressure during the Valsalva manœuvre, and no subsequent "overshoot"; the classical heart-rate changes occurring in normal subjects were also absent (Fig. 2). It would appear, therefore, that in most cases the failing heart is unable to change its stroke volume immediately in response to a rapid fall in filling pressure. Since the pulse pressure remains essentially unaltered, the absence of the "overshoot" is to be expected, if the theory that the latter is due in the normal subject to a decreased pulse pressure is correct. The peripheral constriction in some cases of heart failure must of course modify the response, especially over a short period; but in spite of this an appreciable fall in central venous pressure has been measured.

In the second group of cases there was an increase in pulse pressure during the Valsalva manœuvre, and a subsequent decrease in diastolic pressure, indicating vasodilatation (Fig. 3). It is suggested that these cases, which were clinically the most severe as judged by the height of the venous pressure and course of the illness, were on the falling limb of the Starling filling pressure–output curve, and responded to a decreased filling pressure by an increased stroke output and therefore an increased pulse pressure. The vasodilatation which followed is interpreted as a baroceptor response to the increased pulse pressure, similar to that occurring with other increased pressure transients (Sharpey-Schafer, 1953).

The results are summarized in Fig. 5. In the first group, where no pulse-pressure changes occurred, there was no change subsequently in the diastolic pressure; whereas in the second group the increased pulse pressure was followed by a fall in diastolic pressure, the reverse of the normal.

Discussion

It has been shown previously in animals (Ead *et al.*, 1952) and in man (Sharpey-Schafer, 1953) that increased pressure transients in the circulation give rise to reflex vasodilatation by a baroceptor mechanism. The evidence reported here confirms the theory that, with nervous pathways intact, a decreased pulse pressure produces vasoconstriction and an increased pulse pressure produces vasodilatation. The theory has been shown here to apply not only to the normal but also to the failing circulation. Baroceptor reflexes are active in heart failure provided that the necessary stimulus is applied, as is shown by the cases with increased pulse pressure and subsequent vasodilatation. The lack of baroceptor response in those cases showing no pulse-pressure change is in accordance with this view. Further evidence of baroceptor activity in heart failure in response to pressure changes using other stimuli will be presented elsewhere.

The cases of heart failure reported here were all cases with a raised venous pressure and sinus rhythm. Many other cases of heart disease with a normal venous pressure and patients with abnormal cardiac rhythms have also been investigated, and the results lie on the curve shown in Fig. 5; as might be expected, some are in the normal and some in the pathological part of the graph, with others intermediate in position.

Previous workers (Hamilton *et al.*, 1944; Goldberg *et al.*, 1952; Price and Conner, 1953) have recorded "square wave" patterns on arterial-pressure records in cases of mitral stenosis and occasionally other conditions during the Valsalva manœuvre. The explanations offered have been unsatisfactory, probably because it was not recognized that all forms of heart failure show this phenomenon.

It is of interest that the majority of cases of heart failure did not respond to the acute fall in filling pressure produced during the Valsalva manœuvre by an increased stroke volume, as did the majority of all cases of heart failure studied by the Fick method and venesection (Howarth *et al.*, 1946). The difference in duration of the two sets of observations and the heart-rate changes occurring in the latter group may be sufficient to explain the discrepancy.

Clinical Uses

The main effects of the Valsalva manœuvre may be observed by feeling the pulse in the majority of cases. Rate changes are easily detected, but pulse-pressure changes may also be appreciated. It is usually possible to be certain that the response to the manœuvre is normal by feeling the pulse. Occasionally a spurious "heart failure" response may arise from weak blowing, generally seen in young women who lack the competitive spirit of males and children, or from cheating, which appears to be confined to Royal Air Force personnel who have learned to appose tongue and soft palate, thereby maintaining a mercury column indefinitely without any rise in intrathoracic pressure.

The Valsalva manœuvre may be used to elicit the presence of heart failure in difficult cases and to establish the relative innocence of certain noisy hearts. When used, however, to assess the progress of established cases of heart failure, it shows only too often that little real change has been achieved when they are discharged "compensated" from hospital.

It is also possible by auscultation during the Valsalva manœuvre to decide whether a murmur arises on the right or left side of the heart (Zinsser and Kay, 1950). If the murmur is from the right side it will return immediately on release of the intrathoracic pressure rise; if from the left, a delay of a few beats will be observed. This physical sign is present only if the heart is responding in a normal manner. In most cases of heart failure, since there is no change in stroke output there is no differential accentuation of a murmur. The value of the sign is therefore limited.

Summary

The Valsalva manœuvre causes an acute reduction in the effective filling pressure of the heart and has been used to study the circulation in 62 normal subjects and 63 patients with heart failure. Continuous arterial records showed conspicuous differences in the two groups.

In the normal subjects decrease in arterial-pulse pressure was followed by a rise in diastolic pressure (overshoot). In heart failure from any cause there was usually no change in pulse pressure and no overshoot. In the remaining severe cases the pulse pressure was increased and was followed by a decrease in diastolic pressure.

The results in normal subjects and in heart failure are compatible with the general theory of baroceptor responses to acute pulse-pressure changes. Thus decrease in pulse pressure was followed by vasoconstriction, and increase in pulse pressure by vasodilatation; unchanged pulse-pressure was without effect peripherally.

The Valsalva manœuvre appears to be the simplest repeatable method available at the moment for studying the differences between the normal and failing circulation. It can be used clinically by feeling the pulse.

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VENOUS PULSE IN ATRIAL SEPTAL DEFECT: A CLINICAL SIGN

BY

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The value of observing the venous pulse in the neck is widely recognized, but accurate identification of the waves by timing them against the heart sounds is less commonly practised. This need not be a difficult procedure provided there is no marked tachycardia, particularly where identification of the *a* and *v* waves is concerned, as the former immediately precedes the first heart sound and the latter occurs directly after the second heart sound. Careful observation of the venous pulse in those types of congenital heart disease that predominantly affect the right side of the heart may be of value in establishing the diagnosis. The importance of the large *a* wave in pulmonary stenosis is well recognized (Abrahams and Wood, 1951; Campbell, 1954). It is surprising, therefore, that little attention has been paid to the venous pulse in atrial septal defect. Wood (1950) mentions that it is usually normal, although the venous pressure may be raised in some instances; and Luisada