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VENOUS TONE*

BY

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It is not a new idea that the peripheral venous system can constrict or dilate. The behaviour of the central venous pressure after haemorrhage, in heart failure, or in severe anaemia has long suggested that there are changes in peripheral venous tone and hence in the relative distribution of blood in the venous system. Thus rapid and conspicuous changes in central venous pressure may be observed without any change in circulating blood volume. Strong indirect evidence is provided by consideration of the responses of circulations with and without circulatory reflexes (Sharpey-Schafer, 1956; Sharpey-Schafer and Taylor, 1960). When the reflex pathway is interrupted the circulation behaves mechanically, and mean arterial pressure bears a direct relationship to the arterial pulse pressure and to the stroke output of the heart. It is the time difference between the normal, acting reflexly, and the mechanical, without reflexes, which indicates that in the former the veins, as well as the arteries, must be involved. Thus at the end of the Valsalva manœuvre control pulse pressure is not regained for about 15 seconds in a mechanical circulation; but in the normal, pulse pressure is greater than the control level within five seconds. Similar time differences in stroke output and pulse pressure can be seen in continuous arterial records during other procedures. Graphs of tipping into the erect posture, squatting, or hyperventilation cannot be fully interpreted on the view that arterial constriction or dilatation alone occurs. If, however, it is assumed that the venous system is also responding reflexly, then a fuller analysis is possible.

In the past the main difficulty has been to find methods for the study of peripheral venous tone. Many will be familiar with pressure measurements made in an "isolated" venous segment; but experience with this method over a decade suggests that the results are often more convincing to those who have not used it than to those who have. Modifications of this procedure are better, but are still exposed to serious artifacts.

A method to be described has many advantages in that frequent measurements can be made over short time intervals, and the effect of circulating substances as well as nervous impulses can be studied. In any such investigation there are clear advantages in finding procedures which will dissociate the behaviour of arteries and veins, so that one dilates while the other constricts. No reflex mechanism has yet been found which will do this, but certain circulating substances will.

Isolated Venous Systems

The principle of this method is to measure the pressure in a segment of vein or a system of veins which is isolated from the rest of the circulation. The blood is stationary in these veins, and the only connexion with the rest of the body is through the nervous system. In theory, a rise of pressure indicates venous constriction and a fall of pressure indicates dilatation. In practice, suitable segments of forearm vein occur only in a few individuals. The manometer sensitivity has to be great—about 1 cm. deflection for 1 to 3 mm. Hg. Artifacts are correspondingly increased, and body movement or muscle contraction can be very troublesome. Perhaps the most serious defect is that it is difficult to be certain that small radicals are not entering the inferior aspect of the segment. From a large number of experiments a few satisfactory records have been obtained, and constriction of a venous segment has been demonstrated during the Valsalva manœuvre and on tipping into the erect posture.

A somewhat less difficult modification consists in measuring the pressure simultaneously in the brachial artery and in a large antecubital vein, although high manometer sensitivity is still needed and the consequent artifacts still operate. The limb is cut off from the rest of the circulation by inflating a cuff on the upper arm to a high pressure. After about two minutes the arterial and venous pressures reach a nearly steady level (usually about 20 mm. Hg). A period of 5 to 10 minutes then follows during which experiments can be performed. Eventually the vasomotor nerves become paralysed by the high-pressure cuff. This last effect is often useful, as it is possible to repeat a given manœuvre under nerve-block conditions and thus distinguish artifacts from reflex constriction.

It is a simple matter to show that a rise of pressure occurs in both the arterial and the venous isolated systems after about six seconds' blowing during the Valsalva manœuvre. About 100 experiments of this type have been recorded. The reflex constriction of the arteries previously shown by plethysmography is clearly demonstrated. But it might be argued that if the arteries and veins are still in communication, then the rise of venous pressure might be a passive effect resulting from constriction of the arteries. It can be shown, however, that this is not the case. It is possible to arrange that the pressure in the veins is higher than in the arteries, and yet the same effect is produced. In this experiment two cuffs are placed on the upper arm; by inflating one of these, blood is first collected in the venous system so that the pressure rises to 40 to 50 mm. Hg. The high-

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pressure occluding cuff is then thrown on. With the blood stationary, arterial pressure declines at first rapidly, then more slowly. Venous pressure also falls, for the arterioles are dilating progressively with the ischaemia. (It is shown below that the veins do not dilate from ischaemia.) In most subjects the pressure in arteries and veins is about equal by the time that the experiment can be done. But in a few subjects, probably owing to the arrangement of venous valves, a time may be chosen when the pressure is higher in the veins than in the arteries. A rise of pressure in artery and vein now indicates constriction of both sets of vessels.

The main value of these methods lies in giving a continuous record of the precise time period of changes in arterial and venous tone. Thus an elegant example may be recorded in "long cycle" (60 seconds) Cheyne-Stokes respiration. The constriction of both arteries and veins during hyperpnoea and the dilatation during apnoea proceed as smooth curves until they cease, when the vasomotor nerves become paralysed; incidentally proving that these changes in peripheral vasomotor tone are mediated by the nervous system and not by local changes in arterial gas tensions.

Venous Pressure Increment Per Unit of Inflow

This method depends on the simultaneous measurement of the rate of volume increase and the rate of venous pressure rise while blood is collecting in a forearm. The arrangement is the usual one for plethysmography, with a high-pressure cuff below cutting off the blood-flow to the hand and an upper-arm cuff for applying a collecting pressure. A small needle attached to a capacitance manometer records the pressure in the largest available antecubital vein. If, in fact, the forearm veins are capable of altering their tone, then one would expect that constriction would cause a greater pressure rise per unit inflow and dilatation a lesser.

While blood is collecting in a forearm the volume and the pressure increases describe complex curves, both of which are linear in most subjects over the range used for the measurement of forearm flow. A sigmoid type of pressure curve is not seen if the arm is at heart level, but can be demonstrated if the arm is held vertically above the head and the veins are virtually empty. Under the latter conditions there is an initial flat part to the curve, which then rises more steeply. With the arm at heart level, inflating the collecting-cuff squeezes sufficient blood into the distal forearm veins to increase their pressure to the steeper part of the curve. The measurements are therefore made in the range of 5 to 20 mm. Hg.

A change in venous pressure increment per unit of inflow might be due to alterations in the pressure of surrounding structures rather than to a change in the tone of the venous wall. There is evidence to show that this is not the case. Firstly, in any forearm it is possible to alter the flow through a wide range without altering the venous pressure increment ratio. Short periods of total ischaemia (two minutes) produce, during the subsequent hyperaemia, great changes in flow without any change in the pressure-flow ratio. These results also demonstrate that while short periods of ischaemia dilate the arteries (Lewis and Grant, 1925) the veins are unaffected. Secondly, reflex mechanisms which are known to cause constriction or dilatation of arteries alter the venous pressure-flow ratio in a manner which is abolished when the reflex pathway is

interrupted. Thirdly, it is shown below that circulating substances affect the arteries first, producing considerable changes in flow, and only after an appreciable period arrive at the veins to alter the pressure-flow ratio.

The artifacts of the method bear some resemblance to the well-known artifacts of plethysmography. Thus an initial rapid cuff artifact is common on the pressure curve and may reach formidable proportions if a wide collecting-cuff is used on a fat tapering upper arm. It may be abolished or reduced by using a narrow cuff. In a few subjects, either the volume or the pressure increase or both are non-linear, the volume curve being convex to the left usually, and the pressure curve convex to the right (recording is, of course, from left to right). The pressure increase is most likely to be non-linear if the recording vein is small. Occasionally, too, sudden jumps of pressure may occur in small veins. No calculations can be made from such records.

The results are calculated from the slopes of the venous pressure and forearm volume curves and expressed as $\frac{\Delta P}{\Delta t} / \frac{\Delta V}{\Delta t}$ in arbitrary units of millimetres of mercury per millilitre. Nomenclature has been found to be troublesome. Electrical analogies are not entirely suitable, and terms like "incremental elastance" have been abandoned. It seems better, for the present, to define this ratio as "venous tone" of the forearm. The method has been used to study some 200 subjects.

Reflex Changes in Venous Tone

Posture.—The constriction which has been demonstrated in the arteries on tipping into the erect posture (Brigden *et al.*, 1950) also occurred in the veins (Fig. 1). In both sets of vessels the onset of constriction, mediated through the baroreceptors, was seen to occur after five to six seconds. On tipping back, dilatation occurred more rapidly, often with an "undershoot." These effects were absent in circulations with the reflex system interrupted. In the normal the maximum decrease in arterial pulse pressure on tipping upright is at five

FIG. 1.—Effect of posture on venous tone. Upper graph: normal subject, showing constriction in the erect posture. Lower graph: subject without circulatory reflexes (tabetic), showing no change of tone with alteration of posture.

seconds; thereafter an increase in pulse pressure reflects the constriction of the peripheral venous system, with consequent increase in stroke output.

Valsalva Manœuvre.—This procedure is a more violent method of reducing the filling pressure of the heart. As expected, constriction of the venous system was easily demonstrated in 50 normal subjects (Fig. 2). It can now be seen that the conspicuous "overshoot" in arterial mean and pulse pressures after the Valsalva manœuvre is due to constriction not only of the arteries but also of the veins, thus increasing the stroke output for a number of beats. Changes in venous tone did not occur in subjects with interrupted circulatory reflexes.

Venesection.—Measurements on blood donors venesected 450 ml. showed little change in venous tone; but on increasing the amount of blood removed constriction was demonstrable (Fig. 3). With these large venesections it was noticeable that the skin became pale at about the same time that the veins constricted. These results show that haemorrhage is not just a question of simple reduction of blood volume, nor indeed is estimation of blood volume by itself of much value. Reflex venoconstriction tends to maintain the filling pressure of the heart, and probably explains the well-known discrepancies which often arise between the amount of blood lost and the beneficial effect of a smaller blood transfusion.

FIG. 2.—Venous constriction with the Valsalva manœuvre in a normal subject.

FIG. 3.—Venous constriction with a large venesection in a normal subject.

Coughing.—It has been shown that cough transients imposed on the circulation cause reflex arterial vasodilatation (Sharpey-Schafer, 1953a), and it can now be shown that the venous system also dilates (Fig. 4). Previously, the low pulse and mean arterial pressures which follow a few large cough transients, and which can be of a sufficient order to cause syncope, were explained by reflex arterial dilatation together with some diminution in cardiac output due to the raised intrathoracic pressure which persists for many seconds after coughing has ceased (Sharpey-Schafer, 1953b). This raised intrathoracic pressure, however, is only of the

FIG. 4.—Dilatation of both arteries and veins after coughing.

order of 10 mm. Hg, and it always presented some difficulty that positive-pressure breathing at this level could reduce stroke output to the degree seen in continuous arterial records. Now that the additional factor of reflex dilatation of the veins has been demonstrated, the great reduction in stroke output can be satisfactorily explained.

Ganglionic Blocking Agents

Figs. 5 and 6 show the effect of intravenous tetraethylammonium bromide (T.E.A.B.). There is dilatation of forearm veins, indicating that resting tone, which can be released by blocking the reflex pathway, is present in normal peripheral veins.

It is known that, with the subject strictly supine, mean arterial pressure may fall very little after the intravenous

FIG. 5.—Upper trace: venous pressure. Lower trace: forearm volume. Time marker in seconds. Left-hand record is the control measurement; right-hand record after 200 mg. tetraethylammonium bromide intravenously. The rate of pressure rise compared with rate of volume increase is less after T.E.A.B., indicating venodilatation.

injection of ganglionic blocking agents. On the other hand, tipping into the erect posture will cause arterial pressure to fall to the lowest levels. The problem now is to determine how far the hypotension is due to venous as opposed to arterial factors. In a circulation without reflexes mean arterial pressure bears an approximately linear relationship to the stroke output of the heart, and since it is the veins which govern the latter it would appear that they, and not the arteries, are the major factor. It is also known that in patients with long-standing interruption of circulatory reflexes postural decrease of arterial pressure is slight. This is true both when the afferent pathway is interrupted by diseases such as tabes dorsalis or diabetic neuritis and when the

efferent pathway is chronically blocked, as in long-standing drug treatment of hypertension.

A small number of measurements have shown that in such patients venous tone has returned to normal levels from the subnormal level induced by acute interruption of the reflex system. Reflex responses in the veins, however, are still absent. It would appear, therefore, that there are slow mechanisms, taking weeks or months to operate, which under conditions of blocking may restore normal venous tone. In clinical terms, such a mechanism would explain "recovery" from acute postural hypotension due to disease of the nervous system and the "loss of sensitivity" to adequate doses of hypotensive agents in the treatment of hypertension. It is clear that the usual clinical practice of following such cases by the measurement of arterial pressure by the antiquated cuff method can only be of limited value.

FIG. 6.—Normal supine subject, showing dilatation of both arteries and veins after 200 mg. T.E.A.B. intravenously.

FIG. 7.—Venous tone plotted against total forearm flow. Clear circles—normal subjects; black circles—patients with heart failure. The normal limits lie between 1 and 3 mm. Hg pressure rise per ml. inflow. The cases of heart failure show venoconstriction.

Venous Tone in Heart Failure

It was not originally expected that the method used would give case-to-case information about venous tone as well as changes occurring in any one individual. However, when some 80 normal subjects had been studied, the arbitrary index for resting venous tone in the forearm was seen to lie between 1 and 3 mm. Hg/ml. In all cases of heart failure studied, venous tone was

FIG. 8.—Dilatation of both arteries and veins after intravenous theophylline-ethylene-diamine in a patient with hypertensive heart failure.

increased (Fig. 7). These were patients with hypertensive, ischaemic, and valvular heart disease who showed a "square-wave" response to the Valsalva manoeuvre (Sharpey-Schafer, 1955). The highest degree of venoconstriction was found in severe left heart failure. Some cases of valvular heart disease with great cardiac enlargement and tricuspid incompetence showed only a moderate degree of constriction in the peripheral veins.

The effects of standard clinical treatment were followed in 12 cases. With clinical improvement and a fall of central venous pressure, venous tone decreased towards the normal range. Similar results were obtained in acute experiments, and Fig. 8 shows the effect of theophylline-ethylene-diamine. It has been shown previously that theophylline causes a sharp fall in central

venous pressure as its initial action (Howarth *et al.*, 1947). This is now seen to be due to the effect on the peripheral veins. Intra-arterial theophylline has not yet been given to determine whether or not the venodilatation is a local action.

The high degree of constriction of peripheral veins in heart failure is difficult to explain by arterial baroreceptor mechanisms alone. There is evidence that there is another reflex mechanism from the low-pressure system (auricle or pulmonary veins) on the left side of the heart. Thus in aortic incompetence the great arterial pulse pressure is largely derived mechanically and alters very little as severe heart failure develops, although conspicuous constriction of peripheral veins can be measured. Again, when the central venous pressure of normal subjects is raised by large rapid transfusions, constriction of the peripheral vessels occurs in spite of an increase in arterial pulse pressure. It is the rise of pressure in the left auricle which is the factor common to all these conditions.

Severe Anaemia

Forearm flow is normal or somewhat increased in anaemia. The arterial system therefore tends to be dilated. Fig. 9, however, shows that the venous system

FIG. 9.—Severe anaemia. Venous tone plotted against forearm flow. The solid lines represent the effect of restoring Hb to normal levels. The horizontal dotted lines represent the normal limits (see Fig. 7).

is constricted. When venous tone is plotted against haemoglobin concentration (Fig. 10) it will be seen that the critical level is 40%, and below this figure venoconstriction becomes progressively greater as the haemoglobin falls. The graph bears a striking similarity to that found when cardiac output is plotted against haemoglobin (Sharpey-Schafer, 1944). Indeed, it is probable that venoconstriction is primary and in large part responsible for the increase in cardiac output. As might be expected, there is a relationship between peripheral venous tone and the height of the central venous pressure (Fig. 11). Many would agree, too, that conspicuous pallor of the skin is best seen below 40%

Hb. Since it is venous as opposed to "capillary" or arterial vessels which probably govern the colour of the skin, it is likely that pallor results more from constriction than from any reduction in local haemoglobin concentration.

Beriberi

Two patients with severe beriberi "heart failure" have been investigated. The data from one of these are shown in Fig. 12. This patient was a young alcoholic who clinically, biochemically, and in the response to treatment had classical severe beriberi. On admission,

FIG. 10.—Severe anaemia. Venous tone plotted against Hb%. 40% is the critical level of haemoglobin for venoconstriction.

FIG. 11.—Severe anaemia. Relationship between venous tone and central venous pressure.

with a minute output of 15 litres per minute, there was formidable vasodilatation of peripheral arteries—in terms of forearm blood-flow, about seven times the normal value. At the same time the veins were constricted. The skin was pale and hot, although on

FIG. 12.—Beriberi heart. On admission there is great dilatation of the arteries but constriction of the veins (see text). Venous pressure=central venous pressure.

recovery the patient was found to have a ruddy complexion. With the central venous pressure raised to 25 cm. and considerable cardiac enlargement, a clinical diagnosis of severe heart failure would have been accepted by anyone. However, a record of the Valsalva manœuvre showed that the heart was capable

not only of reducing its stroke volume when the high filling pressure was reduced but of increasing it during the "overshoot," as in the normal subject. In this case the plasma volume change was represented by a rise of 24% Hb on recovery. Thus the high filling pressure driving the heart would appear to be derived from peripheral venoconstriction in the presence of an increase in blood volume.

Perhaps the main interest of the data in severe anaemia and in beriberi lies in the dissociation of effects on the arterial and venous systems. Since arterial pulse pressure is increased, dilatation of both arteries and veins would be expected if reflex influences alone operated. Venous constriction in these conditions, therefore, is more likely to result from humoral mechanisms, but their nature is at present unknown.

Effect of Humoral Agents on Venous Tone

The effects on the venous system of a number of circulating substances have been studied. The results are summarized in the Table and will be published

Table Showing Effect of Humoral Agents on Venous Tone

Substance	Dose	Route	Effect on Forearm	
			Veins	Arteries
L-Adrenaline hydrogen tartrate	25 µg. single shot	I.V.	Constriction	Dilatation
" "	2 µg. min.	I.A.	"	Dilatation followed by constriction
" "	2 µg./min.	I.V., hand veins	"	Nil
L-Noradrenaline bitartrate	0.5 µg./min.	I.A.	"	Constriction
5-Hydroxytryptamine creatine sulphate	10 µg./min.	I.A.	"	Dilatation followed by constriction
Histamine hydrogen phosphate	2 µg./min.	I.A.	"	Dilatation
D-L-Isopropylnoradrenaline hydrochloride	0.1 µg./min.	I.A.	Sl. dilatation	"
Amyl nitrite	5 mg./min.	Inhaled	Dilatation	"
Sodium nitrite		I.A.	"	"

I.V. = Intravenous. I.A. = Intra-arterial.

The effects of these substances on the arterial system are established thus:—Adrenaline: Grant and Pearson, 1938; Holling, 1939; Allen *et al.*, 1946. Noradrenaline: Barcroft and Konzett, 1949. 5-Hydroxytryptamine: Reddie *et al.*, 1955. Histamine: Duff *et al.*, 1953. Isopropylnoradrenaline: Cobbald *et al.*, 1960. Nitrites: Wilkins *et al.*, 1937.

elsewhere (Sharpey-Schafer and Ginsburg, 1961). They provide valuable evidence on the method, for substances introduced into the blood-stream reach the arteries first and only some seconds later reach the veins. Thus it is a simple matter to demonstrate conspicuous changes in forearm blood-flow before any change in the venous-pressure increment ratio occurs, as is well shown by the effects of intravenous adrenaline (Fig. 13).

Three routes of administration are available: intravenous, where the substance passes through the heart and the lungs, and where reflex effects may operate; intra-arterial, where the dose is about 1/50 the intravenous dose and only local effects need to be considered; and local intravenous, where the substance is run into a vein on the back of the hand and reaches part of the venous system being investigated without passing through the arteries or the tissues.

FIG. 13.—Effects of intravenous adrenaline. Upper trace: arterial pressure. Middle trace: forearm venous pressure (identifiable by a small cuff artefact). Lower trace: forearm volume. Small upper record is control. 25 µg. adrenaline injected intravenously as a single shot at black signal on time marker. The change in relative slopes of pressure and volume increments is well shown. The adrenaline has just reached the veins at 22 seconds after injection.

The results have shown that adrenaline, noradrenaline, 5-hydroxytryptamine, and histamine are powerful venoconstrictors; while nitrites and isopropylnoradrenaline dilate the venous system (see Table).

Several substances, such as adrenaline and histamine, have a dissociated action on arteries and veins, dilating the former and constricting the latter.

Summary

A method is described for measuring changes in tone of forearm veins. The rate of venous pressure rise and volume increase in a segment of forearm were recorded simultaneously when a proximal collecting-cuff was inflated. The results were expressed as $\frac{\Delta P}{\Delta t} / \frac{\Delta V}{\Delta t}$ in arbitrary units of mm. Hg pressure per ml. inflow, and defined as "venous tone" of the forearm; an increase in this ratio indicating constriction and a decrease indicating dilatation. The errors of the method and those of confirmatory methods, such as pressure changes in isolated venous systems, are discussed.

Reflex constriction of the normal peripheral venous system occurred on tipping into the erect posture, with the Valsalva manoeuvre, and with large venesections. Reflex venodilatation followed large cough transients. These effects were abolished when the reflex pathway was interrupted. Venodilatation followed the administration of intravenous ganglionic blocking agents, indicating that there is some resting tone in forearm veins.

In heart failure, venous tone was increased, often conspicuously, and fell towards the normal level (1-3 mm. Hg/ml.) on treatment. In severe anaemia, venous tone was also increased though the arteries were moderately dilated; the critical level of haemoglobin was 40%, and the plot of venous tone increase against haemoglobin decrease bore a resemblance to the plot of cardiac output against haemoglobin. In severe beriberi the venous system was constricted in spite of maximal dilatation of the arteries.

Adrenaline, noradrenaline, 5-hydroxytryptamine, and histamine were powerful venoconstrictors. Nitrites dilated the venous system, as did isopropylnoradrenaline.

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THE DANGEROUS VAGINAL PESSARY

BY

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Since January, 1957, 13 patients have been admitted to the Royal Victoria Infirmary with serious pelvic complications associated with the prolonged use of vaginal ring or cup-and-stem pessaries. There have been six cases of primary cancer of the vagina, five of severe chronic ulceration of the vaginal walls, and one case of vaginitis complicated by fulminating pelvic cellulitis, and in one patient a pessary had ulcerated into the bladder and rectum, causing large fistulae; this patient died. Brief case histories are given.

Case Reports

Case 1.—A 55-year-old patient was admitted to the gynaecological ward in January, 1957, with a history of slight irregular vaginal bleeding since the menopause two years previously. During the six weeks prior to admission the blood loss had been continuous. For nine years she had suffered from prolapse, and during this time had worn a vaginal ring pessary which had seldom been changed. On examination she was found to have a crescent-shaped primary cancer of the vagina involving the posterior fornix and the right wall of the vagina. The growth followed exactly the line of contact between the pessary and the vaginal wall.

Case 2.—A 74-year-old widow was admitted to the gynaecological ward in July, 1958, for investigation of post-menopausal vaginal haemorrhage. She had worn a ring pessary for prolapse for over 40 years. She could not remember how often the pessary had been changed, but it had finally been removed by her general practitioner in March, 1958, because of vaginal discharge. On examination she was found to have a horseshoe-shaped cancer of the vagina involving the anterior and both lateral walls. The surrounding tissues were oedematous and infected.

Case 3.—A 66-year-old widow was admitted to the gynaecological ward in February, 1958, for investigation of post-menopausal bleeding. She had worn a ring pessary for prolapse since 1948. To begin with she had changed the pessary regularly, but since 1950 she had left it in the vagina for several months at a time. She periodically douched the vagina, and in January, 1958, noticed that the fluid was blood-stained. She consulted her doctor, who referred her to hospital. Pelvic examination revealed an ulcerating cancer of the vagina involving the posterior fornix and the right upper half of the right vaginal wall.

Case 4.—An 84-year-old woman was admitted to the gynaecological ward in July, 1958, for investigation of post-menopausal bleeding. For 10 years she had worn a ring pessary for prolapse. She had changed the pessary every two or three months but had finally removed it in May, 1958, when she noticed vaginal bleeding. Examination revealed an extensive cancer of the vagina involving the posterior fornix and extending round both lateral walls almost as far as the urethra.

Case 5.—A 75-year-old widow was admitted to the gynaecological department because of vaginal bleeding in October, 1958. For 20 years she had worn a ring pessary for prolapse, and it had been changed every two months by the district nurse. Examination under an anaesthetic revealed a crescent-shaped cancer involving the posterior fornix and right vaginal wall.