EMERGENCIES IN GENERAL PRACTICE

SYNCOPE

BY

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Syncope may be defined as loss of consciousness due to an acute decrease of cerebral blood flow. The common types are seen in everyday life and are caused by normal physiological mechanisms. While the Army holds firm but erroneous views on parade-ground incidents, it is the doctor who must deal with casualties in the audiences of clergymen and schoolmasters, for weakness, anaemia, or epilepsy may still be thought the cause by lay minds. It is well known that environment plays an important part in the common physiological types: thus the whisky drinker in the saloon bar is perhaps more liable to cough syncope than the beer drinker in the public bar, and this syndrome is rarely seen outside big cities with polluted atmospheres. It is worth remembering, too, that the only method of execution which depended on recurrent syncope, crucifixion, was devised in a warm climate. The cold North seems always to have preferred more violent methods.

The rarer forms of syncope often point to disease processes in their early stages—for example, the syncope which occurs on exercise in primary pulmonary hypertension—but they include cases which are hysterical in origin, such as syncope from hyperventilation. It is difficult to take an intelligent history, conduct a proper physical examination, or explain the occurrence to a worried patient without knowledge of the mechanisms involved. In recent years the development of continuous methods for recording rapid circulatory changes in man have made it possible to attempt an analysis of the causes of syncope. The present brief review is based on data available in this department, except where the work of others is indicated in the text. These mechanisms are essentially physiological in nature, and ordinary clinical language, still wedded to morbid anatomy, is inadequate to describe them. The standard

terminology for work on the circulation has therefore been used. For those who are not familiar with the subject a brief list of definitions is given in the footnote.*

Physiology

Cerebral blood flow depends on the supply pressure and the vascular resistance of the cerebral vessels, but the latter is an unimportant factor in syncope except after hyperventilation. With one or two exceptions the supply pressure is virtually the arterial pressure, and when this falls below 50 mm. Hg for a few seconds syncope results. The arterial pressure depends directly on the cardiac output and the total peripheral resistance, and to reach syncopal levels either cardiac output or peripheral resistance must decrease conspicuously or both must decrease simultaneously. In the normal subject, however, these two factors work in opposite directions by means of the baroreceptor reflexes so that within the range of normal living the supply pressure to vital organs, including the brain, is kept nearly constant. Since baroreceptor reflexes are brought into play in every acute change of the circulation, some knowledge of their nature is essential to an understanding of mechanisms causing syncope. There is now evidence that, so far as acute changes are concerned, arterial pulse pressure as well as mean pressure affects the receptors in the carotid sinus, aortic arch, and other regions which are conveniently classified together under the term baroreceptors.

With the nervous system intact increased pulse pressure or large imposed transients (as in coughing) cause peripheral vasodilatation, while decreased pulse pressure causes vasoconstriction. The time of these responses, however, is a little different. While a single large beat or pressure transient can be shown to cause vasodilatation, a single small beat does not cause measurable vasoconstriction. Several beats are needed, and the time, 5 to 6 seconds, may be long enough to result in syncope.

Clinical Varieties

Decreased Filling Pressure and Stroke Output, Baroreceptor Vasoconstriction (Fig. 1)

If a man stands upright the effective filling pressure of the heart falls. In normal subjects the moderate decrease in stroke output in the erect posture is fully compensated by baroreceptor constriction. Syncope will only result where decrease in stroke output is considerable.

A rise of intrathoracic pressure (Valsalva manœuvre) causes an acute decrease in the filling pressure of the heart by trapping blood behind closed venous valves. The degree and rate of decrease depend on the height of intrathoracic pressure rise and the previous state of vasomotor tone. In conditions of vasodilatation, therefore, a moderate Valsalva manœuvre may cause syncope, as when straining at stool after getting out of a warm bed. Powerfully built middle-aged men may cough so violently and continuously that intrathoracic pressure is raised to 200 to 400 mm. Hg for many seconds. This is the commoner of the two mechanisms causing cough syncope. It is probable that syncope in whooping-cough and in breath-holding in infants has a similar cause, though continuous records of the two latter conditions are not yet available.

*Definitions.—Supply pressure is arterial pressure minus venous pressure. Baroreceptor reflexes used to be called baroreceptor reflexes and before that carotid sinus and aortic reflexes. As the word implies, they are reflexes sensitive to pressure change. The exact anatomical sites of all those vascular end-organs which send afferent impulses to the central nervous system is unknown in man. The efferent pathway from vasomotor centre to peripheral arterioles is via the sympathetic nervous system. Pressure transient is a pressure change lasting less than a second. It includes a single heart beat, a cough, a sneeze, and jumping from a height on to a hard floor. These changes can be imposed on top of the arterial pressure. Effective filling pressure is the pressure which distends the ventricles of the heart just before systole. It is often regarded as the same as the venous pressure but is a better term when large changes in intrathoracic pressure may be involved. This pressure and the rate are the main factors which determine cardiac output. Ectopic rhythm connotes auricular flutter, paroxysmal auricular or ventricular tachycardia, or auricular fibrillation. Stroke output is the output of the heart with each beat. Minute output is the output of the heart during a minute; owing to changes in heart rate, changes in stroke output are not necessarily in the same direction as changes in minute output. Valsalva's manœuvre is a rise of intrathoracic pressure lasting 10 seconds or more. It is performed by forced expiratory effort against an obstruction, such as a closed glottis, a closed mouth and nose (A. M. Valsalva, *De aure humana tractatus*, p. 84, Utrecht, 1707), or a mercury column, and occurs in everyday life when straining at stool, lifting heavy weights, singing, or blowing long notes, and when the chest wall is compressed by external agencies.

Increased Filling Pressure, Decreased Stroke Output, Baroreceptor Vasoconstriction (Fig 2)

The simplest example occurs when the heart stops completely, as in a Stokes-Adams attack. Baroreceptor vasoconstriction in such attacks is shown by the greatly raised arterial pressure or "overshoot" when the heart starts again.* Very similar in mechanism is the onset of a rapid ectopic rhythm, usually above 180 beats a minute, in a damaged heart. The decrease in output is so great that

FIG. 1

FIG. 2

FIG. 1.—Syncope from Valsalva manœuvre and continuous coughing. Notation in this and subsequent diagrams is as follows: B.P.=arterial blood pressure. C.O.=cardiac output. V.P.=effective venous filling pressure. T.P.R.=total peripheral resistance. Control period before first vertical line. Mechanisms preceding syncope between vertical lines. Syncope after second vertical line. FIG. 2.—Syncope from Stokes-Adams attack, rapid ectopic rhythms, myocardial infarction, pulmonary embolism, exercise in pulmonary hypertension, exercise in normal subjects.

arterial pressure cannot be maintained in spite of intense baroreceptor constriction. It is probable that syncope with acute myocardial infarction and massive pulmonary embolism have a similar mechanism, though continuous records have not yet been obtained in these events.

An important though rare form of syncope is seen in primary pulmonary hypertension and occasionally in other conditions affecting the right heart. It occurs during or shortly after exercise. Acute right ventricular failure is present; the filling pressure rises steadily and cardiac output falls until it reaches a level where compensatory constriction is unable to maintain arterial pressure above 50 mm. Hg.† During syncope the heart slows. Patients have been known to die in the attack.

It is possible, though unproved, that the normal right heart might fail in a similar manner. Athletes and soldiers are known to have syncope and even die under conditions of prolonged maximum effort. The untrained public school-boy running for his house is perhaps the greatest risk, since the mental stimulus to above maximum effort is then more formidable than in any other known circumstance. Cases are known where acute left ventricular failure has followed such effort. Some normal human hearts, however, may fail on the right side before the left. Unfortunately there are no physical signs directly after acute right heart failure and no characteristic post-mortem findings if death occurs.

Decreased Filling Pressure and Stroke Output, No Baroreceptor Response (Fig. 3)

It has already been explained that arterial pressure while standing is maintained by baroreceptor constriction. Absence of this defence mechanism is therefore likely to jeopardize cerebral blood flow and consciousness. Theoretically, baroreceptor reflexes may be blocked at any point in the pathway. On the efferent side the effect of the various ganglionic

*Barlow, E. D., and Howarth, S. (1953). *British Medical Journal*, 2, 863.

†Howarth, S., and Lowe, J. B. (1953). *Brit. Heart J.*, 15, 47.

blocking agents or extensive sympathectomies are well known. Not only is syncope apt to occur in the erect posture, but any procedure such as the Valsalva manœuvre which decreases the filling pressure and stroke output may have the same result. It should be remembered, however, that if the heart is failing an acute decrease in filling pressure will not reduce stroke output but may even increase it. Cases of hypertension commonly treated by these methods show less postural syncope than normal subjects, since it

FIG. 3

FIG. 4

FIG. 3.—Syncope from ganglionic blocking agents, sympathectomy, and disease of the afferent nervous system. FIG. 4.—Syncope from the erect posture after squatting (A) and intermittent coughing (B).

can be shown by proper methods that a proportion of hypertensives have heart failure when first seen.

Recently it has been possible to show that in certain diseases of the nervous system there is a complete block of afferent baroreceptor impulses. Syncope is rather uncommon in such cases, although the erect arterial pressure is of the order of 80/60 mm. Hg. But if some additional factor such as a hot environment is present, syncope is very easily induced. The data on these cases will be published shortly.

Baroreceptor Vasodilatation, Decreased Filling Pressure, and Stroke Output (Fig. 4)

If a normal subject squats blood is pressed out of the veins of the legs and cardiac filling pressure rises. The increased stroke output and pulse pressure cause a baroreceptor vasodilatation. On standing erect suddenly the hydrostatic fall of filling pressure and cardiac output takes place while this vasodilatation is still present. For a few seconds, therefore, arterial pressure may reach syncopal levels. There can be few individuals, especially gardeners, who have not experienced this form of syncope on occasion.

Coughs are imposed on the vascular system and have two effects: they stimulate the baroreceptors producing reflex vasodilatation, and they are followed by a rise of intrathoracic pressure. Since this post-tussive rise of intrathoracic pressure is invariable in all types of subject—normal, diseased, and denervated—it is thought to be mechanical rather than reflex. Its effect is to reduce cardiac filling pressure and, if the heart is normal, the stroke output. If cough transients are large, as in strongly built middle-aged men, syncope may result.

Other Forms of Vasodilatation, Decreased Filling Pressure, and Stroke Output (Fig. 5).

Hyperventilation, by decreasing arterial $p\text{CO}_2$, causes vasodilatation in muscle vessels by an action on the central nervous system and thence via the vasomotor nerves. It

also causes cerebral vascular constriction. Any subsequent procedure, such as assumption of the erect posture or the Valsalva manoeuvre, which reduces filling pressure and stroke output may decrease arterial pressure to a level (less than 80 mm. Hg) which will cause syncope. Owing to the cerebral constriction the critical level is higher than in other forms of syncope.

Although limb blood flow is increased about threefold by indirect heating of normal subjects, cardiac output is also increased and syncope does not occur. In complete

FIG. 5

FIG. 6

FIG. 5.—Syncope from hyperventilation or exercise (A) and indirect heating or carotid sinus pressure (B). FIG. 6.—Syncope from vasovagal faint reflex resulting from haemorrhage, erect posture, supine posture in pregnancy, spinal anaesthesia, positive-pressure breathing, apomorphine, and psychological causes.

thyroid deficiency, however, the initial low cardiac output does not rise on heating and limb flow may increase tenfold. The arterial pressure commonly falls to syncopal levels. Patients with myxoedema are known to collapse in hot baths.

The procedure of carotid sinus pressure causes peripheral vasodilatation and inhibition of the heart beat. In older subjects syncope may easily be produced. Since the fashion in male collars has become less rigid, self-induced syncope of this type has become rarer.

Violent muscular effort results not only in extreme vasodilatation of muscle vessels but in a conspicuous increase in cardiac output. Normally, therefore, arterial pressure is maintained. It is possible, however, that the erect posture without movement directly after maximal effort may sometimes result in a discrepancy between these two factors leading to syncope.

It must be admitted, however, that the occasional collapse of athletes after the finishing post is still obscure, owing to lack of data.

Decreased Filling Pressure and Stroke Output, Acute Peripheral Vasodilatation (Vasovagal Reflex) (Fig. 6).

The common faint involves a reflex mechanism which can apparently overcome the baroreceptor response. The efferent effects are the same however fainting is produced; there is an acute vasodilatation in muscle vessels. The afferent part of this reflex is still uncertain, but the following facts have to be explained: (1) there is a decrease in cardiac filling pressure before muscle vasodilatation occurs; (2) cases of heart failure never faint; (3) the reflex is abolished by anaesthesia; and (4) it is not known to occur in species other than man.

At the present time the most attractive theory is that the afferent nerve endings lie in a ventricle, probably the left, and that they are stimulated by large pressure transients which develop during systole when virtually all the blood has been ejected and ventricular muscle is contracting on an empty chamber. Such large transients have been shown

to occur under special but similar conditions in animals.* All normal humans can be made to faint provided enough blood is removed. Many of the other methods which cause fainting can be shown to be the same mechanism. Thus standing erect on a hot day is "bleeding" into the veins of the lower part of the body. The supine posture in late pregnancy causes obstruction of the inferior vena cava by the uterus. The erect posture after spinal anaesthesia results in a conspicuous decrease in cardiac filling pressure, and prolonged positive-pressure breathing has the same effect.

The morphine derivatives, particularly apomorphine, will cause this form of syncope. The first event seems to be a decrease in cardiac filling pressure, suggesting that splanchnic dilatation precedes firing of the faint reflex. It is very possible that spontaneous or psychological fainting has a similar mechanism to apomorphine, for any form of nausea causes a decrease in cardiac filling pressure. It is known that acute muscle vasodilatation occurs in this common condition, but the events which precede the reflex are still unrecorded.

The idea that cortical activity and the efferent sympathetic pathway are alone involved in psychological fainting seems untenable if it is remembered that in spite of all the needles that are thrust at them patients with heart failure never faint.

Multiple Mechanisms and Obscure Forms of Syncope

From what has been said above it is clear that two or more mechanisms may be combined. Thus squatting and hyperventilating followed by rising to the erect posture and performing the Valsalva manoeuvre (Fig. 7) uniformly produce syncope in normal subjects.

In two forms of vasovagal fainting the preceding events are still uncertain. A proportion of normal subjects breathing low oxygen mixtures lose consciousness with a low blood pressure, bradycardia, and vasodilatation in muscle vessels.† The injection of 3 litres of air into the rectum in the erect posture will also produce the same result, and some persons faint easily when given enemas. It is possible here that a reflex splanchnic vasodilatation precedes the faint reflex.

Cases of aortic stenosis not uncommonly have syncope on effort. Observed clinically the arterial pressure is below 50 mm. Hg and there is bradycardia. But there is no evidence of acute left heart failure such as dyspnoea and rales in the lungs. A mechanism similar to that illustrated in Fig. 2 but occurring on the left side of the heart is therefore unlikely. It is tempting to believe that with the increase in heart rate blood cannot be ejected through the stenosed valve without large systolic pressure increases in the cavity of the left ventricle, and that these pressure transients are sufficient to fire the vasovagal faint reflex.

A form of carotid sinus syncope without fall in arterial pressure was described in 1933 by Weiss and Baker.‡ At

*Henry, J. P., and Gauer, O. H. (1950). *J. clin. Invest.*, **29**, 855.

†Anderson, D. P., Allen, W. J., Barcroft, H., Edholm, O. G., and Manning, G. W. (1946). *J. Physiol. (Lond.)*, **104**, 426.

‡Weiss, S., and Baker, J. P. (1933). *Medicine (Baltimore)*, **12**, 297.

FIG. 7.—Syncope from multiple mechanisms. Squatting and hyperventilation between vertical lines 1 and 2, erect and performing Valsalva manoeuvre between vertical lines 2 and 3. B.R.=brain resistance.

that time continuous records were not available and extensive search since suggests that this syndrome does not exist. Other terms that might now be dropped include Gowers's syndrome, Nothnagel's syndrome, and Charcot's laryngeal vertigo. Those modern neurologists who continue to believe in the last must produce continuous records in support of their claim.

Diagnosis

Although the history is often decisive in differentiating syncope from vertigo, epilepsy, or minor cerebral lesions if there have been multiple attacks of unconsciousness, a single attack may present great difficulties. There is one sign which is helpful if a reliable person has witnessed an attack. If arterial pressure falls below 50 mm. Hg for more than a few seconds the posterior pituitary is stimulated, secretes its hormone, and causes pallor of the skin which persists for many minutes after syncope. There is also diminished excretion of urine for several hours, but it is uncommon to obtain a clear-cut history of this effect. The most important differential diagnosis is from epilepsy. It cannot be stressed too strongly that a full epileptiform convulsion is common in quite normal individuals if a syncopal attack is severe and prolonged. A history of injury and loss of sphincter control is therefore less useful than the textbooks would have one believe. The crucial measurement during the attack is the arterial pressure; this is increased in epilepsy and is, of course, greatly decreased in syncope. If the attack is observed from the beginning it may be possible to assess the changes in arterial pressure by ordinary clinical methods, but these changes may be very rapid and are easily missed. In difficult cases it is often necessary to attempt to reproduce the attack while continuous measurements are made. The following case illustrates this point.

A strongly built middle-aged bus driver had attacks of dizziness and occasional unconsciousness when his head or trunk were twisted into certain positions. He claimed he went red in the face during the attack and was pale afterwards. This story seemed meaningless, and no further light was thrown by observation of an attack, except that his claims appeared to be true. Continuous arterial records were obtained in a bent-forward posture with and without an attack. It became clear that he had acquired the unfortunate habit of closing his glottis in certain postures and then straining. The rise of intrathoracic pressure was 150 mm. Hg or more. This formidable Valsalva manoeuvre was sufficient to reduce arterial pressure to syncopal levels on occasion.

Treatment

Once the mechanism of a particular form of syncope is understood, treatment consists in avoiding the circumstances which cause the attack. On hot days soldiers should not stand to attention for long periods, gardeners should sit, not squat, and pregnant women should not lie supine on hard couches. Subjects susceptible to vasovagal attacks, a tendency which is often inherited, should be needed supine rather than sitting or erect.

The most difficult problem is presented by those patients whose syncopal attacks result from bad habits such as hyperventilation or violent coughing. Explanation or, better, demonstration of the nature of the attack may be successful, but too often the patient has other personality difficulties and the habit remains. Occasionally more drastic measures may be necessary, as in the following case.

A powerful man aged 55 had cough syncope of a severe nature. On occasion intrathoracic pressure reached 450 mm. Hg for many seconds. Syncopal attacks occurred 9 or 10 times a day and caused injury and danger in traffic. Ordinary methods were unsuccessful, so tracheotomy was performed. With a tube open to the atmosphere the patient still achieved 200 mm. Hg intrathoracic pressure for some seconds and eventually coughed out his firmly fixed tube. Because the patient was incapacitated prefrontal leucotomy was considered justifiable. This broke the habit and a year later he was at work.

Prognosis

The ability to faint by one of the physiological mechanisms indicates that the circulation is normal, and the patient should be told this firmly. Indeed, life assurance

companies might well regard this ability in elderly persons with some gloom, for it would mean that they have not got heart failure and might well live beyond the average time. The only risk lies in the environmental circumstances. Thus a steeplejack may fall off a building; a motor-car driver may kill himself or, worse still, other people; a competitive swimmer may hyperventilate before diving, then strain, have syncope, and so drown; while doctors who leap from hot baths to answer the telephone may injure themselves severely. Very rarely, now that crucifixion is abolished, the upright posture is forcibly maintained after syncope and may be one of the causes of death in panic-stricken wedged crowds. In general, as soon as the victim is flat on the ground recovery takes place. In those forms of syncope associated with heart disease the prognosis is more ominous. The severity of the underlying heart lesion is, of course, the decisive factor. The risk of death during an attack is often as uncertain as the risk of a second myocardial infarction, extending over periods of five minutes to twenty years.

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A MEDICAL SCHOOL FOR WESTERN AUSTRALIA

BY

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The foundation of a new medical school within the British Commonwealth of Nations is a rare event, and so will always make medical news. But where the circumstances of the foundation are as unorthodox as they are courageous, and where this courage and this heterodoxy succeed immediately beyond all optimistic anticipation, the story assumes a fascination that merits its telling beyond that local sphere where communal pride in achievement keeps the progress of the venture ever before the public eye.

Western Australia

Of all the States of the Commonwealth of Australia, Western Australia is the largest, but it is also the most thinly populated. At the end of the last war its total population did not exceed half a million. The last ten years have seen that population increase by more than a third. In part this increase has been due to a persistent high birth rate and a low infant mortality, but it derives also from a forward-looking immigration policy which has brought to the State many useful citizens, "new Australians" from Britain and from a variety of European countries. It is anticipated that the population will continue to increase and will pass the million mark in the seventies of this century. Inevitably the major area of growth will be in and immediately around Perth, the capital city, in which region more than half of the population are likely to continue to live.