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ORIGINAL ARTICLE

Low pressure hydrocephalus and ventriculomegaly: hysteresis, non-linear dynamics, and the benefits of CSF diversion

M. S. LESNIAK, R. E. CLATTERBUCK, D. RIGAMONTI & M. A. WILLIAMS*

*Departments of Neurological Surgery and *Neurology, Johns Hopkins Hospital, Baltimore, Maryland, USA*

Abstract

Low pressure hydrocephalus (LPH) is a rare clinical condition. We report our experience with 10 patients treated at the Johns Hopkins Hospital. We reviewed the records of 10 patients (five men, five women; mean age 43 years) treated between 1996 and 2000. All underwent intracranial pressure (ICP) monitoring and subatmospheric cerebrospinal fluid (CSF) drainage with an intraventricular or lumbar catheter. All patients developed ventriculomegaly: five following aneurysmal subarachnoid haemorrhage; one after meningitis; one after intraventricular haemorrhage. Three patients presented with chronic aqueductal stenosis. Ventriculomegaly was clinically detected on average 12 days after presentation. Mean ICP was 4.8 mmHg (range 0–10). All patients improved only in the setting of negative pressure CSF drainage, and were subsequently treated with low pressure ventriculo- or lumboperitoneal shunts. At 1 year, eight patients (80%) showed good recovery to minimal disability; seven patients (70%) had resolving ventriculomegaly. The mechanism of low pressure hydrocephalus remains unclear. In our cohort, different aetiologies were responsible for the change in compliance/elastance of the brain parenchyma and subsequent development of ventriculomegaly. We propose that while ventriculomegaly (and therefore neuronal dysfunction) can be initiated in the setting of high ICP, the maintenance of ventriculomegaly at normal or low ICP is a physiological example of hysteresis. This behaviour, which has been characterized by the chaos theory of non-linear dynamics as a Hopf bifurcation, explains how a system can exhibit two different states (ventricular size) at a single parameter value (ICP). Most importantly, it helps to explain how lowering ICP in the setting of LPH can resolve ventriculomegaly and its neurologic sequelae.

Key words: *Cerebrospinal fluid shunts, hysteresis, low pressure hydrocephalus, non-linear dynamics, subarachnoid haemorrhage, ventriculomegaly*

Introduction

The syndrome of normal pressure hydrocephalus (NPH) was first described by Hakim and Adams in 1965.[1,2] At the time they described three patients in their 60s who had in common progressive dementia, abnormal gait, urinary incontinence and bilateral Babinski signs. Serial measurements of lumbar CSF pressure in these patients showed no evidence of elevation and a pneumoencephalogram in each case showed a dilated ventricular system. The placement of a shunt resulted in an immediate and dramatic improvement in their neurological status.

Since this first description of NPH, several case reports have described the syndrome of low pressure hydrocephalus (LPH).[3–5] This syndrome is distinct from NPH and characterized by ventriculomegaly in the setting of very low ICP. It may follow subarachnoid haemorrhage, meningitis, parasitosis or head trauma. The usual symptoms are headaches, nausea, vomiting and lethargy; however, it may also present with the cognitive and neurological symptoms of patients with normal pressure hydrocephalus. In isolated cases, these symptoms have been shown to respond to CSF diversion; however, very little data exist to support the theory that shunting these patients offers any long-term benefit.

We report our experience with 10 patients treated at the Johns Hopkins Hospital during the period between 1996 and 2000. With the exception of three individuals, all of our patients developed ventriculomegaly following subarachnoid haemorrhage, intraventricular haemorrhage or meningitis. In spite of very low CSF pressure, all 10 patients eventually received a ventriculo- or lumboperitoneal shunt, and showed a significant improvement in their neurological status.

Correspondence: M. S. Lesniak, Department of Neurological Surgery, Johns Hopkins Hospital, 600 N Wolfe Street, Meyer 8–161, Baltimore, Maryland 21287, USA. Tel: (410) 955–7482. Fax: (410) 502–5768. E-mail: mlesniak@jhmi.edu

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Methods

We retrospectively reviewed the charts of all patients admitted to the Neurosciences Critical Care Unit (NCCU) who subsequently developed ventriculomegaly in the setting of low intracranial pressure. Patients were graded according to the Glasgow Coma Scale (GCS) at the time of admission, neurological decline and, again, following CSF drainage. Outcome was assessed by determining the Glasgow Outcome Scale (GOS) and neurological status (Karnofsky score) at the time of discharge, and after 1 year of follow-up care.

Diagnostic evaluation in all patients included insertion of either an intraventricular catheter or a lumbar catheter for ICP monitoring. A therapeutic trial of continuous external CSF drainage was performed utilizing the methods established for patients with NPH.[6–9] Briefly, each patient underwent external CSF drainage by adjusting the position of the CSF drip chamber to achieve a CSF drainage rate of 10 ml/h. ICP was then measured every 15 minutes with the patient remaining in supine position. These patients were then assessed over the course of 3–7 days to determine whether there was any improvement in their neurological condition. Of note, the CSF profile was analysed every 48 h. The endpoint was defined either as improvement in neurological status or completion of CSF drainage for the period of the full 7 days. Patients who unequivocally improved with CSF drainage subsequently received a permanent ventriculoperitoneal (PS Medical Delta Valve, Minneapolis, MN) or lumboperitoneal (Cordis Horizontal-Vertical Lumboperitoneal Valve, Miami, FL) shunt.

Statistical significance was determined by the Kruskal–Wallis non-parametric analysis of variance followed by the nonparametric analog of the Newman–Keuls multiple comparison test.[10]

Results

Patient demographics

We identified 10 patients admitted to the Johns Hopkins Hospital between January, 1996 and June, 2000 who developed ventriculomegaly and low intracranial pressure. There were five women and five men who ranged in age from 23 to 70 years. Of the 10 patients, five developed ventriculomegaly following aneurysmal subarachnoid haemorrhage. In the other five, the aetiologies were meningitis (1), intraventricular haemorrhage (IVH, 2) and aqueductal stenosis (3). None of the patients received prior brain irradiation or had a history of cerebral infarction, which would account for the ventricular enlargement. The clinical and radiological detection of hydrocephalus occurred on average 12 days after initial presentation. The clinical profiles of all 10 patients are summarized in Table I.

ICP monitoring and CSF drainage

Despite low ICP (range 0–10 mmHg; group mean 4.8 mmHg), all patients underwent a therapeutic CSF drainage trial, which was achieved by lowering the CSF drip chamber to a point at which the CSF drainage was at least 10 ml^2/h (Fig. 1, Table II). This goal was achieved only at negative CSF pressures. We observed no concomitant changes in arterial blood pressure. Significant improvement in symptoms and neurological status was documented by clinical examination in all patients (Fig. 2; $p = 0.001$). This improvement was evident as early as 12 h after initiating the drainage, and occurred in all within the first 72 h. Depending on the pressure at which the neurological symptoms improved, each of the patients subsequently received a low pressure shunt (Delta 1.0 if improvement seen at ICP > -5 mmHg; Delta 0.5 for improvement seen at ICP ≤ -5 mmHg). One patient (case 5), who was initially treated with a Delta 0.5 valve, continued to show neurologic deterioration and progressive ventriculomegaly. This patient underwent a revision in which a Mischler reservoir without a valve was connected to the ventricular catheter and distal tubing. In two other patients (cases 1 and 3), the family opted for the placement of a lumboperitoneal shunt. In those cases, the Cordis horizontal-vertical lumboperitoneal shunt (vertical closing pressure 170–240 mmH_20) was selected.

TABLE I. Patient demographics

Case	Age (year)	Sex	Atiology	Admission GCS	Day of decline after admission	Symptom of decline
1	70	F	SAH	13	17	↑ Lethargy
2	53	F	SAH	12	12	Worsening headache
3	43	F	SAH	12	6	Failure to follow commands
4	53	M	SAH	7	18	Flexor posturing
5	49	M	SAH	6	24	Extensor posturing
6	34	M	IVH	8	15	↑ Lethargy, confusion
7	28	F	Meningitis	11	17	↑ Lethargy, confusion
8	52	M	AS	10	7	↑ Lethargy, new seizures
9	23	M	AS	8	10	Stupor, extensor posturing
10	24	F	AS	13	3	Worsening headache

SAH, subarachnoid haemorrhage; IVH, intraventricular haemorrhage; AS, aqueductal stenosis.

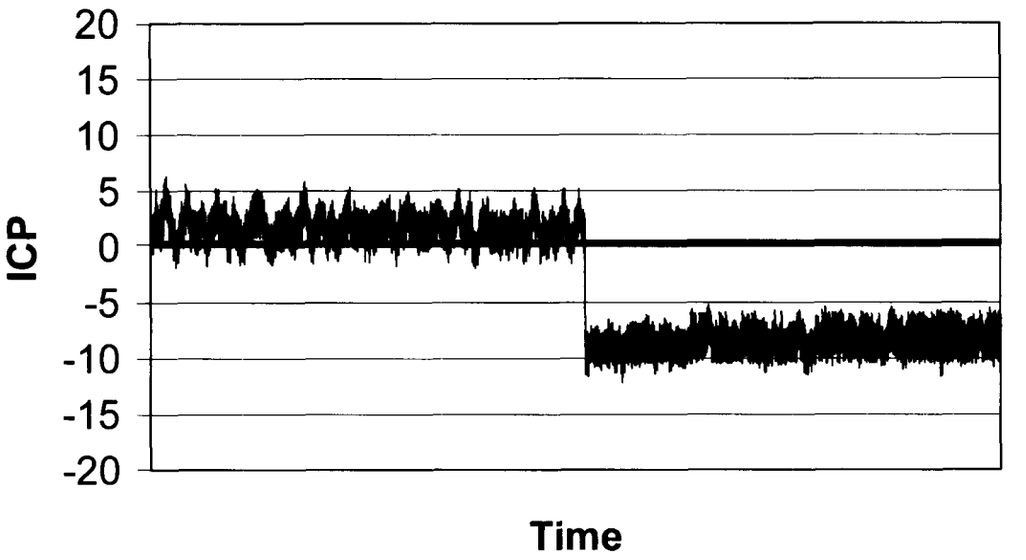

FIG. 1. ICP during monitoring and diagnostic drainage. The waveform was obtained by measuring ICP before and after CSF drainage in a representative patient (case 10). In spite of low pressure, this patient complained of a severe and excruciating headache that was unrelated to position and not relieved by medication. After a period of overnight CSF drainage, the symptoms completely resolved and the patient was pain free.

TABLE II. Intracranial pressure monitoring and shunt selection

Case	Opening pressure at admission (mmHg)	ICP at initiation of CSF drainage (mmHg)	Mean ICP at which symptoms improved (mmHg)	Selected shunt
1	26	10	–2	LPS-LP
2	27	8	–3	Delta 1.0
3	30	5	–2	LPS-LP
4	37	4	–5	Delta 0.5
5	29	3	–8	Reservoir
6	33	2	–7	Delta 0.5
7	26	6	–5	Delta 0.5
8	25	4	–5	Delta 0.5
9	27	5	–3	Delta 1.0
10	31	1	–7	Delta 0.5

LPS-LP: Cordis horizontal/vertical low pressure lumboperitoneal shunt, Reservoir: Mischler reservoir.

TABLE III. Outcome by GOS and neurological status at 1 year

Case	GOS	Karnofsky score	Complications/ revisions
1	GR	90	None
2	GR	100	Shunt revisions (obstruction × 1, infection × 1)
3	MD	80	None
4	SD	50	Shunt revisions (obstruction × 4, infection × 1)
5	SD	40	Shunt revisions (obstruction × 1, infection × 1)
6	MD	80	Shunt revision × 1 due to obstruction
7	GR	90	Shunt revision × 1 due to obstruction
8	GR	90	Shunt revision × 1 due to obstruction
9	GR	100	Shunt revisions (obstruction × 1, infection × 1)
10	GR	100	Shunt revisions (obstruction × 6, infection × 2)

GR, good recovery; MD, moderate disability; SD, severe disability; VG, vegetative state, Dead.

Follow-up

Follow-up ranged from 1 to 3 years. All patients had significant improvement in neurological function at one year (Table III). Of note, all but two patients experienced a shunt obstruction or infection requiring a revision during the first year. These shunt obstructions were likely related to the high protein content (average 350 mg/dl; range 199–1300 mg/dl) in the CSF of patients with SAH, IVH or meningitis. In turn, multiple shunt revisions due to obstruction may have posed the greatest risk for subsequent shunt infections.

Seven patients had radiologic evidence of resolving ventriculomegaly (Fig. 3A).

Discussion

In this study, we describe a group of patients with very low intracranial pressure and ventriculomegaly who are very different from the majority of hydrocephalic or shunt-dependent patients. In the setting of subarachnoid haemorrhage, infection or trauma, the signs and symptoms of low pressure hydrocephalus developed relatively slowly over several days to weeks. In all of the cases, slowly deteriorating mental status, along with increasing lethargy and

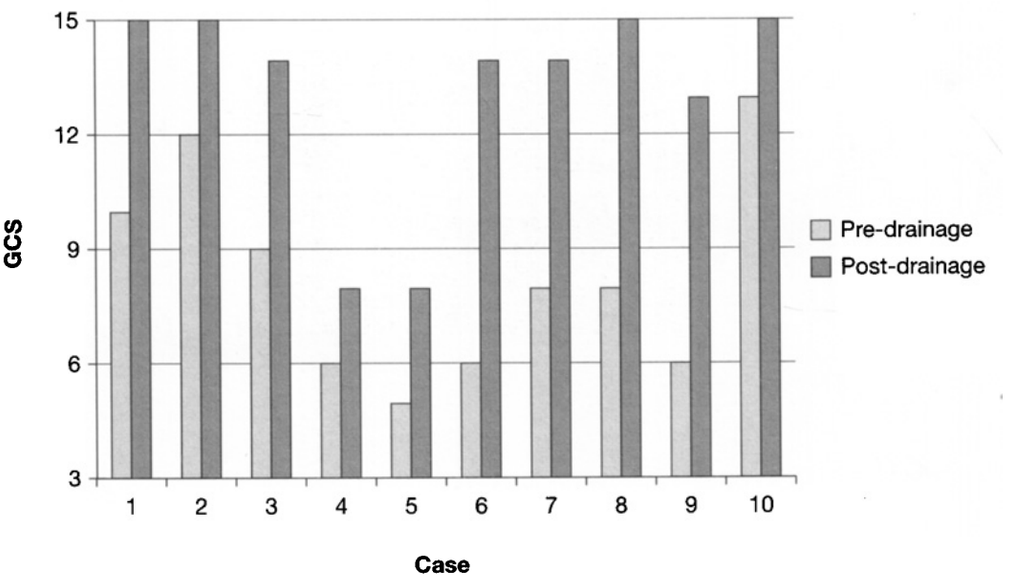

FIG. 2. GCS before and after therapeutic CSF drainage. The GCS was obtained at the time of neurological decline in the setting of ventriculomegaly. Following CSF drainage, the GCS was again assessed and unequivocally improved in all cases ($p = 0.001$).

progressive obtundation, were the hallmark findings. Radiological findings were consistent with marked ventriculomegaly. Measurement of CSF pressure either via lumbar or ventricular catheter showed the mean pressure to be below or within the normal range. Nevertheless, all of the patients showed significant neurological improvement in the setting of negative pressure CSF drainage trial. Based on these findings, all of the patients were treated with low pressure shunts to further optimize their neurological recovery.

Monro–Kellie doctrine and hysteresis in LPH

The Monro–Kellie doctrine provides a fundamental understanding of the relationship between intracranial pressure and volume, and helps to explain the phenomenon responsible for low intracranial pressure in the setting of ventriculomegaly. According to the doctrine, the cranial vault is a closed system containing the brain parenchyma, cerebrospinal fluid and blood. An increase in any one of these must be offset by an equal decrease in another or else ICP will rise. In this system, the compliance of the brain dV/dP, as well as elastance dP/dV, govern the relationship between intracranial pressure and volume. In cases of acute hydrocephalus or shunt obstruction, small incremental changes in intracranial volume are accompanied by sustained, high ICP waves.[11] These elevated ICP waves, in turn, are associated with many of the acute symptoms, including headaches, nausea, vomiting and drowsiness.[12]

In contrast to this classic presentation of hydrocephalus with elevated ICP, patients with low pressure hydrocephalus have a more insidious course and presentation. All of the patients in our study showed a very slow, though progressive, deterioration of neurological function, as manifested by decreasing levels of consciousness and neurological function over several days. Moreover, all of these patients showed significant enlargement of their ventricular system in spite of low to normal intracranial pressure. To explain this apparent incongruity, we refer to Figure 3B which shows the relationship between intracranial pressure and volume in two separate states (one as ICP increases, the other as it decreases). Initially, as ICP increases secondary to any increase in intracranial volume, it does so along curve 1. However, acute distention of the ventricular system changes the compliance/elastance relationship in the cranial vault, shifting the curve to the right (curve 2). Indeed, previous work by Lofgren and Allen, suggested that pressure-volume curves can shift in the context of any pathophysiological process.[13–15] As a result, when ICP later decreases with CSF drainage, it reaches a lower value, but only at a much larger ventricular volume. This increase in compliance/decrease in elastance of the central nervous system (CNS) parenchyma is the mechanism responsible for the development and maintenance of ventriculomegaly. Even lower ICP (i.e. negative pressure) is then required to reduce the ventricles to normal size. This quality of a system in which a single value of a parameter such as ICP can correspond to two separate system behaviours of states (i.e. ventricular volume) is called hysteresis. It suggests a dynamic mathematical behaviour known as a subcritical Hopf bifurcation, which governs the coexistence of two attractors at the same parameter value (see the Appendix). Such a phenomenon is not likely to be unique to the CNS and has been reported in models that explain the mechanical properties of other organs, such as vocal cords, aorta, lungs and the cardiac muscle.[16–19]

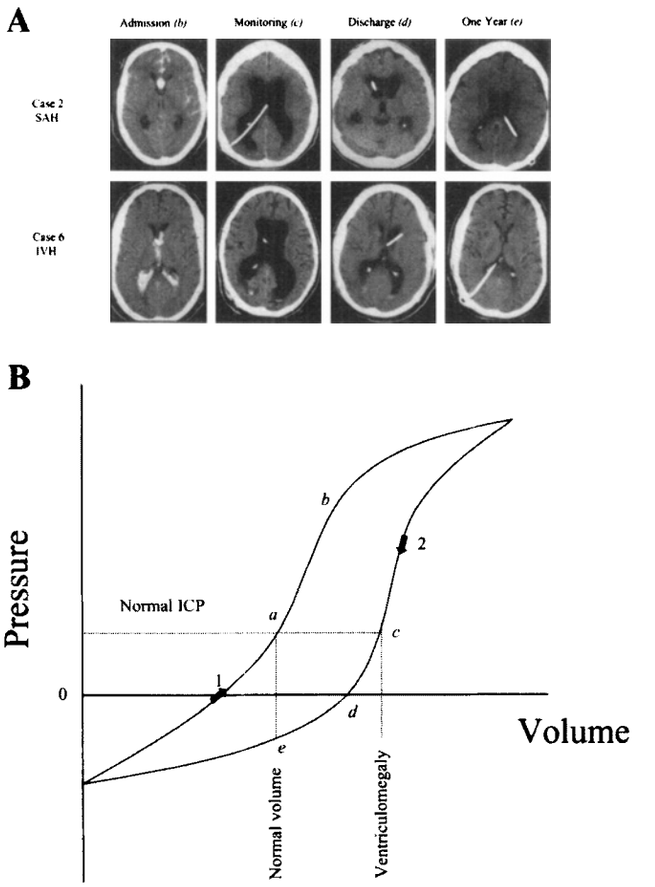

FIG. 3. Hysteresis and low pressure hydrocephalus. (A) Imaging characteristics of low pressure hydrocephalus. These brain computed tomography (CT) scans illustrate the clinical progression of two representative patients in the study at (*b*) admission, (*c*) monitoring, (*d*) discharge, and (*e*) one year. (B) Hysteresis. As ICP increases with any increase in ventricular volume ($a > b$), it does so along curve 1. Acute distention of the ventricular system increases the compliance of the cranial vault, shifting the curve to the right (curve 2). When ICP subsequently decreases with CSF drainage, it reaches a lower value at a much larger ventricular volume (*c*). Even lower CSF pressure is then required to return the ventricles back to normal size ($d > e$). This quality of a system in which a single value of a parameter such as ICP can correspond to two separate system behaviours (i.e. CSF volume) is called hysteresis. It suggests a dynamic mathematical behaviour known as a subcritical Hopf bifurcation which governs the coexistence of two attractors at the same parameter value.

The increase in compliance/decrease in elastance of CNS parenchyma may be a result of several predisposing factors. First, any condition that causes atrophic changes in the parenchyma, such as trauma, haemorrhage, ischaemia, infection or radiation may also result in a more compliant brain. These changes are analogous to those in the senescent brain, where a relatively low packing density in the white matter predisposes to NPH.[4,20] Secondly, patients with long-standing hydrocephalus, such as individuals with aqueductal stenosis may suffer from the loss of lipid and protein and develop periventricular gliosis, which might collectively alter the brain elasticity when their hydrocephalus is subsequently treated.[4,21–24] Finally, the presence of a craniotomy and/or craniectomy performed at surgery may increase brain compliance by decreasing the rigidity of the cranial vault, and thus changing the inherent relationship between intracranial pressure and volume. In summary, almost any insult to the central nervous system that is capable of altering the cranial vault, the brain parenchyma or the flow of CSF, can result in marked dilatation of the ventricular system, consistent with ventriculomegaly and paradoxically low ICP.

Abnormalities of ventricular size and CSF flow have long been known to cause neurological symptoms. The signs and symptoms of hydrocephalus have traditionally been associated with increased ICP. Neural tissue, however, is known to withstand high compressive stresses, such as those associated with increased ICP as long as they are applied equally in all directions.[25,26] It is not unreasonable, therefore, to assume that as long as the ventricles remain dilated, white matter tracts are stretched, generating neuronal dysfunction. In the case of NPH, the descending prefrontal white matter tracts may be stretched out as they travel around the ventricular system, accounting for the prominent gait apraxia. Indeed, Yakovlev *et al.*, first proposed this theory to explain

spastic paraplegia of hydrocephalic patients.[27] On the other hand, prolonged high ICP can also lead to neuronal damage and death. The sequence of events leading to axonal damage consists of ventriculomegaly, followed by disruption of the periventricular ependyma, periventricular oedema, axonal destruction, secondary myelin disintegration and finally reactive astrocytosis.[22] In studies of adult cats, hydrocephalus most severely affected the corpus callosum, as well as the cortical mantle, leading to a paucity of myelinated fibres.[21] In the same study, however, early shunting resulted in prompt reversal of ependymal disruption and periventricular oedema. Moreover, it was associated with restoration in the thickness of the cortical mantle, which may explain the relationship between hydrocephalus and mental function.

Therapeutic intervention

In our study cohort, continuous therapeutic CSF drainage below atmospheric pressure was accompanied by a gradual improvement in the patient's neurological function. Each of the patients subsequently received a CSF shunt, the profile of which was based on the mean pressure at which the neurological condition improved. For patients requiring extremely low to subatmospheric pressure, we utilized Delta 1.0 and 0.5 valves, respectively. This strategy ultimately led to neurological improvement in all patients and gross radiological evidence of resolving ventriculomegaly in seven. It is possible that with continued treatment, a further decrease in the size of the ventricular system may occur in the remaining three patients.

Taken together, the 10 patients in our study suggest that once ventriculomegaly is established, the ventricular pressure has to be lowered dramatically before the radial stress is reduced. Theoretically, this may allow the stretched axonal fibres to return to their normal length. In the states of ventriculomegaly accompanied by low pressure hydrocephalus, it is not the pressure, but rather the stretching of the fibres around the ventricular surface that is most likely responsible for the patient's neurological symptoms. By diverting CSF from the enlarged ventricles, one can promote the patient's recovery to previous neurological function.

Conclusions

The development of low pressure hydrocephalus and ventriculomegaly may occur following any intracranial event, such as subarachnoid haemorrhage, intraventricular haemorrhage, meningitis or in the setting of aqueductal stenosis. In this study, we propose that while ventriculomegaly can be initiated in the setting of high ICP, the maintenance of ventriculomegaly at normal or low ICP is a physiological example of hysteresis. This behaviour helps to explain not only how a system can exhibit two different states (i.e. ventricular size) at a single parameter value (ICP), but also how lowering ICP in the setting of LPH can resolve ventriculomegaly and its neurological sequelae.

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Appendix

In the theory of nonlinear dynamic systems, the qualitative change of a dynamic behaviour at a critical value of a parameter is called a bifurcation.[28–30] At a Hopf bifurcation, an equilibrium position changes its stability and an oscillation (limit cycle) is generated.

Two types of Hopf bifurcations are possible. We illustrate them in the figures below, where we represent the dynamic behaviour as a function of a control parameter, a. In the figures, a solid line represents stable equilibrium (a position of a limit cycle), and a dashed line represents unstable equilibrium. At the supercritical Hopf bifurcation (Fig. A1), as the parameter increases, a stable equilibrium position bifurcates into an unstable position and a stable limit cycle. The supercritical Hopf bifurcation is seen as a smooth transition in which the formerly stable equilibrium starts to wobble in extremely small oscillations that grow into a family of stable periodic orbits as the parameter changes.

In the subcritical Hopf bifurcation (Fig. A2), as the parameter increases, a stable equilibrium position and unstable limit cycle coalesce into an unstable equilibrium position. A subcritical Hopf bifurcation is seen experimentally as a sudden jump in behaviour. As the parameter a is increased in the figure, the system follows the equilibrium $v = 0$ until reaching the bifurcation point $a = 0$. After passing this point, no stable equilibrium exists and the orbit is immediately attracted to the only remaining stable orbit, an oscillation of large amplitude. In this case, the bifurcating path of periodic orbits of the Hopf bifurcation exists for $a < 0$. The periodic orbits thrown off from the origin for negative a are unstable orbits. For $a < 0$, they provide a basin boundary between the attracting equilibrium and the attracting periodic orbit on the outside.

An important nonlinear effect often seen in the presence of a subcritical Hopf bifurcation is hysteresis. To illustrate this concept, assume a system is being observed and shows a particular behaviour at a given parameter setting. Next, the parameter is changed and then returned to the original setting. Now, the system displays a behaviour completely different from the original behaviour. This change in a system behaviour, which depends on the coexistence of two attractors at the same parameter value, is called hysteresis.

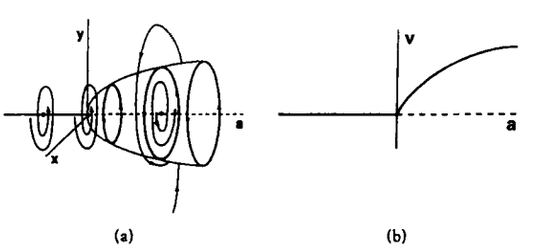

FIG. A1. Supercritical Hopf bifurcation. (a) The path $\{(a, 0, 0)\}$ of equilibria changes stability at $a = 0$. A stable equilibrium for $a < 0$ is replaced by a stable periodic orbit for $a > 0$. (b) Schematic path diagram of the bifurcation. Solid curves are stable orbits, dashed curves are unstable. [Reprinted with permission from Allgood *et al.*[31]]

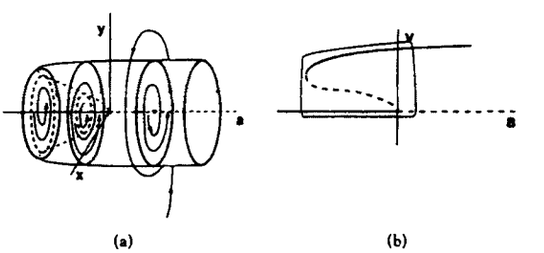

FIG. A2. A subcritical Hopf bifurcation with hysteresis. (a) There is bifurcation at $a = 0$ from the path $r = 0$ of equilibria. At this point, the equilibria go from stable to unstable and a path of unstable periodic orbits bifurcates. The periodic orbits are unstable and extend back through negative parameter values, ending in saddle node at $a = -1$. An additional path of attracting periodic orbits emanates from the saddle node. (b) Schematic diagram of bifurcation paths. The rectangle shows a hysteretic path. The vertical segments correspond to sudden jumps. [Reprinted with permission from Allgood *et al.*[31]]