

# The small intestine and irritable bowel syndrome (IBS): a batch process model

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## Abstract

Control faults in a batch process model of the small intestine create the symptoms of all types of Irritable Bowel Syndrome. The model has three sequential processing sections corresponding to the natural divisions of the intestine. A brain controller with four sub-controllers, each with a unique neuro-transmitter, governs it.

Each processing section has a sub-controller to manage transport. Sensors in the intestinal walls provide input, and output goes to the muscles lining the walls of the intestine. The output controls the speed of food soup, moves it in both directions, mixes it, controls the absorption rates of nutrients, chemicals & water, and transfers it to the next section at the correct speed (slow).

The fourth sub-controller manages the addition of bile. It obtains input from the first section of the process via the signaling hormone Cholecystokinin and sends output to the muscle that empties the gall bladder. The correct amounts of bile are then added to the first section.

The transport sub-controllers produce output only when input is received. When output is missing, the enteric nervous system applies a 'default' condition called the Migratory Motor Control (MMC). This condition is normally active only when there is no food in the intestine. But if food is present and a transport sub-controller fails to provide output, then the MMC moves the food soup to the end of that section. The movement is in one direction only (forward), at a speed dependent on the types of food eaten, and environmental factors affecting the autonomic nervous system.

When the speed of the MMC is 'too fast',  
the symptoms of Irritable Bowel Syndrome are produced.

## Introduction

This hypothesis evolved from observations of a case of IBS-D over a period of decades. Examination found no visible physical faults in the patient's digestive system. The progressive worsening of the disorder was consistent with the gradual loss of neuro-transmitter(s) in the brain. Cases of IBS-C & A also provided evidence. Dietary trials were carried out.

## Explanations

Here you will find background information necessary to understand the hypothesis:

### *Autonomic nervous system (ANS)*

This is the interconnected networks of nerves that control automated systems in the human body. Activity levels in these networks increase when adrenal hormones are released on arising in the morning, and when stress occurs. After the initial morning peak of activity, a relaxation may occur. Levels go to a minimum overnight.

### *Bicarbonate*

This is made by the pancreas and walls of the small intestine. It neutralizes stomach and food acids so that the pH of food soup is neutral to slightly alkaline. This is necessary because human digestive enzymes do not work in acid conditions.

### *Borborygmii*

These are loud gurgling sounds that come from the abdomen. They are common in some types of IBS, and are caused by liquid food soup moving at high speed in the small intestine.

### *Bile*

This is made in the liver, stored in the gall bladder, and secreted into the duodenum. Bile emulsifies fats into tiny droplets that can then be digested by lipase enzymes.

### *Cholecystokinin (CCK)*

When food soup is pumped from the stomach into the duodenum, CCK peptide hormones [3] are released into the bloodstream. One travels to the brain and stimulates the sub-controller for the gall bladder. This then contracts the muscle that moves bile into the duodenum.

### *Enteric nervous system (ENS)*

This is a division of the autonomic nervous system (ANS). It is the network of nerves that controls the automated functions of the digestive system.

### *Duodenum*

This is the first processing section of the small intestine, and it is about 25 centimetres long.

### *Glucose-dependent insulinotropic peptide (GIP)*

This hormone is released from the duodenum when food soup is pumped in from the stomach. It stimulates the release of insulin hormone from the pancreas and slows gastric emptying.

### *Gall Bladder*

A storage organ for bile produced in the liver. Small amounts of bile are continually released into the duodenum from the gall bladder. Some foods also cause the release of small amounts of bile. Larger amounts are released by a muscle under the control of the brain.

### *Ileo-cecal valve (ICV)*

This valve terminates the small intestine. Normally closed, it opens when adrenal hormone levels are elevated. This happens on arising in the morning and when stress occurs.

### *Ileum*

This is the third and last processing section of the small intestine. It is between 4 and 6 metres long.

### *Insulin*

A hormone made in the pancreas that moves glucose into cells.

### *Irritable Bowel Syndrome (IBS)*

This is a common digestive disorder that has three main types; IBS-C (constipation predominant), IBS-D (diarrhoea predominant), and IBS-A (alternating constipation & diarrhoea). The author has identified a fourth type and has named it IBS-B (bile deficient). Rates of occurrence have been measured at 5-25% in 'Western' populations. The financial burden for the USA alone has been estimated at tens of billions of dollars yearly [1, 2].

### *Jejunum*

This is the second processing section of the small intestine. It is between 2 and 3 metres long.

### *Large intestine (colon)*

This two metre long muscular tube accepts food soup from the small intestine and prepares it for release via the anus.

### *Liver*

This is an organ that manufactures bile (from cholesterol), and stores it in the gall bladder.

### *Migratory motor control (MMC)*

When there is no food in the small intestine, brain control is switched off, and the ENS applies a peristaltic wave called the MMC that sweeps the intestine slowly from start to finish. But if food is present and the brain fails, then the MMC moves it instead.

### *Neuro-transmitters*

These are chemicals that the nervous system uses to transmit signals between nerve cells. There are a considerable number of them, and many are unknown to science.

### *Pancreas*

An organ that, when stimulated by hormones, delivers enzymes and bicarbonate into the duodenum. It also manufactures insulin hormone.

### *Secretin*

This hormone is released from the duodenum when food is pumped in from the stomach. It stimulates the pancreas to release enzymes and bicarbonate into the duodenum, and slows gastric emptying.

### *Small intestine*

This is a narrow flexible muscular tube that accepts food soup from the stomach, digests it, and then moves it into the large intestine. It is about 6-9 metres long and divided into three sections (in order); duodenum, jejunum, and ileum.

## The Hypothesis

Digestion in the small intestine is a batch process, with three sequential sections corresponding to the natural divisions of the intestine; the duodenum, the jejunum and the ileum. Processing is governed by a brain controller divided into four sub-controllers, each equipped with a unique neuro-transmitter. Control faults in this process cause the disorder; Irritable Bowel Syndrome.

### *The batch process*

The duodenum is the first section. It accepts acidified food soup from the stomach and releases CCK, GIP & Secretin into the bloodstream. Bile from the gall bladder, enzymes & bicarbonate from the pancreas, are mixed into the soup. Acids are neutralized and fats are emulsified, then the soup is moved slowly to the next section.

The jejunum is the second section. It accepts slowly moving food soup from the duodenum. The soup is mixed so that nutrients are digested and efficiently absorbed at the correct rate. It is then moved slowly to the next section.

The ileum is the third section. It accepts slowly moving food soup from the jejunum. The soup is mixed so that bile salts and enzymes can be reabsorbed and returned to the gall bladder and pancreas. Soup is then conditioned by removing some water, and moved into the colon through the ileo-cecal valve (ICV).

### *Process control*

There are two levels of control;

The *primary* level is a brain controller. This is divided into four sub-controllers, each with a unique neurotransmitter. Three sub-controllers manage transport and mixing, and the fourth controls the addition of bile. The sub-controllers produce output only when input is received.

The three transport sub-controllers accept input from sensors in the walls of the intestine. The sensors respond to the amount of food soup and the amount and type of insoluble fibre present. If food is absent the sub-controllers remain switched off.

Output from the transport sub-controllers manages the movement and mixing of food soup in the intestine. Correct control happens regardless of the variable input caused by different foods. Soup is mixed by moving it backwards and forwards. It is then transferred to the next section at the correct speed (slow).

The fourth sub-controller adds the correct amount of bile to the food soup. When food is pumped in from the stomach, cells in the wall of the duodenum release peptide hormones (Cholecystokinins or CCK), into the bloodstream. One of these hormones travels to the brain where the fourth sub-controller detects it, then outputs a signal to the muscle that empties the gall bladder.

A *secondary* level of control is provided by the ENS. It manages transport with the MMC. This normally operates only when primary control

and food soup, are absent. However if soup is present when primary control is missing, then the MMC moves the soup. The speed is dependent on;

1. The amount and types of insoluble food fibre.
2. Food selection, food preparation, and timing of eating.
3. Environmental and behavioural factors that affect the autonomic nervous system.

There is no control of mixing or timing, and transport is in one direction only (forward). When the speed of the MMC is 'too fast', IBS symptoms occur.

The ENS also adds small amounts of digestive chemicals when it detects; the amount and types of fibre eaten, cooked proteins (meats, fish, & eggs), fruit acids (alpha-hydroxy acids), dairy proteins, and some herbs & spices (e.g. ginger). However the amount added is normally insufficient for complete digestion.

#### *Control Faults*

1. One or more of the four unique neuro-transmitters in the primary controller may be deficient or absent. This reduces or eliminates output from the controller.
2. A toxic insult may destroy intestinal sensors that secrete hormones, and provide input to the primary controller. This can reduce the amounts of bile, enzymes, and bicarbonate entering the intestine, and reduce or eliminate output from transport sub-controller(s).
3. Surgical procedures may sever nerves connecting the primary controller and the small intestine.

4. Misalignment of neck vertebrae may put pressure on nerves connecting the brain and the small intestine.
5. In pregnancy and childbirth, there may be pressure or damage to nerves connecting the small intestine and the brain.
6. In infancy, development of nerve connections from the brain to the small intestine, may fail to be completed.
7. Any other fault that interrupts communication between the;
  - a. Brain, small intestine & gall bladder.
  - b. Duodenum and pancreas.

#### *The 'IBS Barrier' - Constipation*

A barrier is created when food soup is present in the small intestine, and a section governed by the secondary transport controller (MMC) precedes a section controlled by a primary transport sub-controller. When the MMC moves food soup 'too fast' then the primary sub-controller constricts the intestine to stop the fast flow. It is programmed only to accept soup that travels 'slowly' at the correct speed. The 'IBS Barrier' causes the symptoms of bloating and constipation.

The Barrier is caused by parts of the ANS, so variation in the level of activity in this system causes the Barrier to change its strength. It is strongest on arising in the morning when adrenal hormones are released to kick-start the metabolism. The symptoms of the Barrier are at their worst when breakfast is eaten. They increase in strength again when stress releases adrenal hormones during the day, and decrease when the ANS returns to a low level of activity.

### *Diarrhoea*

This symptom occurs when the terminating section of the process (ileum) is under the control of the MMC. When food soup is moved too soon & 'too fast' into the colon, it contains high levels of acids, bile & enzymes, and has not been conditioned correctly. The colon is automatically evacuated by the ENS when this insult occurs.

The ileo-cecal valve (ICV) at the end of the small intestine is not under brain control. Its state is changed by the level of activity in the ANS. When adrenal hormones are released on arising, the valve is easy to open, and a defective ileum immediately pushes its contents into the colon (the morning rush).

When stress releases adrenal hormones during the day, the valve is again easier to open. Overnight the valve becomes firmly closed when the autonomic nervous system relaxes. When IBS is severe however, the ileum can push food soup through the ICV at any time.

Diarrhoea may also occur when the ileum transport sub-controller is not defective, for these reasons;

1. Insufficient bile is added to the duodenum and the small intestine contains undigested fat.
2. Insufficient bicarbonate is added to the duodenum and the small intestine becomes acidic.

*The following sections explain what happens when neuro-transmitter levels are deficient in one or more of the four brain sub-controllers...*

### *IBS-B (bile deficient IBS)*

When the output from the fourth brain sub-controller is missing or deficient, then insufficient bile salts are added to the food soup. Undigested fats will impair nutrient uptake in the jejunum, reabsorption of chemicals in the ileum, and cause diarrhoea.

Indigestion is followed by a fast, loose, greyish bowel movement containing fat (steatorrhea). The absence of the brown bile pigment stercobilin causes the grey colour, and when fat enters the colon, the ENS evacuates it. IBS-B may occur alone, but often it accompanies one of the other types of IBS. When it does, all symptoms become severe.

### *IBS-C (constipation predominant IBS)*

There are six forms of IBS-C;

1. *The duodenum sub-controller output is deficient or missing;* this causes an 'IBS Barrier' to form at the start of the jejunum. A breakfast of cereal immediately triggers severe bloating in the upper abdomen. Back-pressure in the duodenum keeps the valve from the gall bladder & pancreas closed, and insufficient digestive chemicals are added. If the Barrier persists then constipation occurs. If it relaxes soon after breakfast then undigested fat will cause diarrhoea next morning (as for IBS-A Form 1).
2. *Form 1 together with IBS-B.* Symptoms are severe. If the Barrier persists constipation occurs. If it relaxes quickly then diarrhoea happens when food reaches the end of the ileum, or on arising next morning (as for IBS-A Form 2).

3. *The jejunum sub-controller output is deficient or missing*; this causes an 'IBS Barrier' to form at the start of the ileum. A breakfast of cereal triggers borborygmii followed by hard-to-detect, slight to moderate bloating in the mid-abdomen. If the Barrier persists then constipation occurs. If it relaxes soon after breakfast then constipation & diarrhoea do not occur.
  4. *Form 3 together with IBS-B*. Symptoms are severe. If the Barrier persists then constipation occurs. If it relaxes soon after breakfast then undigested fat will cause diarrhoea (similar to IBS-D Form 4).
  5. *The duodenum & jejunum sub-controller outputs are deficient or missing*; this causes an 'IBS Barrier' to form at the start of the ileum. A breakfast of cereal triggers borborygmii followed by hard-to-detect, slight to moderate bloating in the mid-abdomen. If the Barrier persists then constipation occurs. If it relaxes soon after breakfast then constipation & diarrhoea do not occur.
  6. *Form 5 together with IBS-B*. Symptoms are severe. If the Barrier persists then constipation occurs. If it relaxes soon after breakfast then undigested fat will cause diarrhoea (similar to IBS-D Form 6).
2. *Form 1 together with IBS-B*. Symptoms are severe. Borborygmii may start soon after breakfast. Diarrhoea occurs when food soup reaches the end of the ileum, or on arising next morning.
  3. *The ileum & jejunum sub-controller outputs are deficient or missing*; when food soup reaches the jejunum a short time after breakfast starts, borborygmii begin. Diarrhoea occurs on arising next morning.
  4. *Form 3 together with IBS-B*. Symptoms are severe. Borborygmii start soon after breakfast. Diarrhoea occurs when food soup reaches the end of the ileum, or on arising next morning.
  5. *The ileum, jejunum & duodenum sub-controller outputs are deficient or missing*; a breakfast of cereal triggers immediate borborygmii. Diarrhoea occurs on arising next morning.
  6. *Form 5 together with IBS-B*. Symptoms are severe. Borborygmii start when a breakfast of cereal begins. Diarrhoea occurs when food soup reaches the end of the ileum, or on arising next morning.

*IBS-A (constipation & diarrhoea)*

There are two forms of IBS-A;

1. *The duodenum & ileum sub-controller outputs are deficient or missing*; this causes both IBS-C Form 1 and IBS-D Form 1 together. Constipation and diarrhoea alternate irregularly. The state of the ANS controls the alternation.
2. *Form 1 together with IBS-B*. Both IBS-C Form 2 and IBS-D Form 2 occur together. Symptoms are severe.

*IBS-D (diarrhoea predominant IBS)*

There are six forms of IBS-D;

1. *The ileum sub-controller output is deficient or missing*; when food soup reaches the ileum several hours after breakfast, borborygmii begin. Diarrhoea occurs on arising next morning.

### *Summary*

The 4 types and 15 forms of neuro-transmitter deficient IBS, created from combinations of defects in the four sub-controller outputs, are presented in Table 1.

### *Other Causes of IBS*

The part(s) of the intestine that are affected will not be as defined as they are when a neuro-transmitter is deficient or missing. If nerves or motor sensors are damaged, symptoms will be similar but with timing differences. If sensors that release CCK are damaged, IBS-B occurs. If sensors that release GIP are damaged, symptoms similar to type II diabetes may occur. If sensors that release Secretin are damaged, the small intestine becomes acidic, and diarrhoea occurs.

### *Importance of the theory*

It explains;

- The cause of the common digestive disorder Irritable Bowel Syndrome.
- How to reduce IBS symptoms.
- How the autonomic nervous system controls the digestion.
- That cereals & legumes are the wrong foods for humans to eat.

### *Current theories of IBS*

Some are;

- Bacterial overgrowth
- Over-regulation of the small intestine by the brain.
- Food allergies.

None explain why there are multiple types and forms of IBS, with constipation or diarrhoea or both, and why borborygmii, bloating, and cramping occur. This theory explains how all symptoms are created and points to under-regulation by the brain being the cause of most types of IBS.

### **Evaluation of the hypothesis**

Supporting evidence:

#### *No apparent damage*

Medical examination of most IBS patients shows no visible damage to the small intestine. The problem is likely to be in its control systems.

#### *Cereal & Legume Fibre*

Diets that remove cereals and whole legumes [4, 5] greatly reduce symptoms. Over-stimulation by the insoluble fibre in these foods causes 'too fast' speeds when the secondary controller (MMC) is regulating transport.

#### *Stress*

This triggers and aggravates IBS symptoms showing that the disorder is likely to involve the autonomic nervous system.

#### *Difficulty digesting fats*

This theory identifies five causes;

1. Severe bloating causes back-pressures in the duodenum preventing the release of sufficient bile salts, bicarbonate, & enzymes.
2. Continual diarrhoea will cause a total loss of bile & enzyme stores.
3. Defective chemical sub-controller fails to release sufficient bile from the gall bladder (IBS-B).
4. Damage to duodenal sensors that release CCK hormone (IBS-B).
5. Damage to duodenal sensors that release Secretin hormone. Now insufficient bicarbonate is added to the small intestine, it becomes acidic, and fats are not digested.



### *Diarrhoea*

IBS D & A patients can have high levels of protease enzymes in their fast bowel movements. These enzymes attack the skin around the anus, causing irritation & scarring. They are not being recycled by the ileum before food soup is discharged into the colon. This is an ileum control problem.

The 'morning rush' is a characteristic symptom of IBS. A fast, loose bowel movement occurs soon after arising. This is when levels of adrenal hormones peak and the valve at the end of the small intestine (ICV) becomes easy to open. The ileum immediately pushes its entire contents prematurely through the ICV. High levels of acids, enzymes & fats cause the ENS to rapidly evacuate the colon.

### *Progressive onset*

An IBS-D patient started to suffer symptoms as a teenager. Symptoms were intermittent until about age 40, when they began to increase in frequency. At age 45, they were present every day, and at age 55, there appeared to be no control of the small intestine left. This progression is symptomatic of a gradual loss of neuro-transmitter(s) in the brain.

### *Intestinal bloating*

Bloating with associated cramping can start when breakfast is eaten. Stress during the day can trigger it again. Overnight it usually disappears.

The ANS is at a high level in the morning, high in response to stress and low overnight. It is likely to be causing the bloating.

The symptom of bloating displays two degrees. It is either severe in the

upper abdomen, or slight to moderate and hard-to-detect in the mid-abdomen;

- The duodenum is short (25 centimetres), so when the 'IBS Barrier' is at the start of the jejunum and the stomach continues to pump in food soup, bloating is severe in the upper abdomen.
- The jejunum is longer (2 to 3 metres), so when the 'IBS Barrier' is at the start of the ileum, it causes slight to moderate bloating in the mid-abdomen that is hard-to-detect.

### *Intestinal cramping*

- Cramping associated with bloating is caused by the MMC trying to push food soup through an 'IBS Barrier' created by a primary transport sub-controller. The force used depends on the type of food eaten, and the state of the ANS.
- Cramping associated with borborygmii is the MMC moving food soup 'too fast' in the small intestine.
- Cramping followed by diarrhoea, occurs when food soup moves prematurely into the colon. The soup may contain acids, enzymes & fats, and this insult causes the ENS to rapidly evacuate the colon.

### *Visual hallucinations*

Bile is made in the liver from cholesterol, stored in the gall bladder, inserted into the duodenum to emulsify fats, and later it can be reabsorbed in the ileum and recycled back to the gall bladder.

The ileum transport sub-controller manages this recycling process. When the body's free cholesterol level is low, all bile salts are recycled. When the body's free cholesterol level is high, bile salts are allowed to escape in the stool.

When the ileum transport sub-controller is defective, bile salts can no longer be recycled and they are lost in the stool. This creates a shortage of cholesterol. Now the liver needs to make more bile salts, and it has to requisition cholesterol from the brain. This causes visual hallucinations (kaleidoscopic moving patterns of light in the field of vision).

A cholesterol excess can occur when IBS-B is present, or when back-pressure in the duodenum prevents the release of bile. The gall bladder becomes full, and cholesterol cannot be reduced by making more bile salts. Instead it has to be moved into the brain and this also causes visual hallucinations.

### Evidence against the hypothesis

All the IBS symptoms observed by the author, fit neatly into the hypothesis, but the number of cases studied is limited. Many IBS subjects reported that their symptoms varied somewhat from the model. But differences in age, constitution, diet, lifestyle, disease state, and cause, mean that there is wide variation in expression of symptoms.

Since this is a hypothesis, some or all of it may be wrong. However it should serve to direct researchers into areas they have not yet visited.

### Testing the hypothesis

Clinicians may be able to confirm the existence of the two 'IBS Barriers' predicted by the hypothesis for neurotransmitter deficient IBS-C;

1. '*IBS Barrier*' at the start of the *jejunum*; the key symptom is immediate severe bloating in the upper abdomen on eating breakfast. If the Barrier persists, constipation results. If the Barrier relaxes quickly after breakfast, there is no constipation, and instead borborygmii & diarrhoea may occur (IBS-C Forms 1 & 2 and IBS-A Forms 1 & 2).
2. '*IBS Barrier*' at the start of the *ileum*; key symptoms are borborygmii soon after starting breakfast, then slight to moderate bloating in the mid-abdomen that is hard-to-detect. If the Barrier persists, constipation results. If the Barrier relaxes quickly after breakfast, symptoms may subside (IBS-C Forms 3 & 5), or borborygmii & diarrhoea may occur (IBS-C Forms 4 & 6).

To find a hard-to-detect 'IBS Barrier' in the mid-abdomen, measurement of mid-abdominal diameter should be made before & after breakfast. The presence of cramping in the mid-abdomen, signals that a Barrier is occurring.

The two 'IBS Barriers' may be able to be detected by X-ray scanning the abdomen after a low fat breakfast that contains cereal fibre (not corn) and a signaling compound.

IBS-D may display three variations of the symptom of borborygmii. If digestive chemicals have been exhausted by continual diarrhoea, this test will not be possible:

1. *The ileum transport sub-controller is defective;* borborygmii start several hours after breakfast (IBS-D Form 1).
2. *Both the ileum and jejunum transport sub-controllers are defective;* borborygmii will begin a short time after starting breakfast (IBS-D Form 3).
3. *All transport sub-controllers are defective or IBS-B is present;* borborygmii will begin almost immediately after starting breakfast (IBS-D Forms 2, 4, 5, 6).

Researchers studying brain function can look for the four small intestine brain controllers, with their unique neuro-transmitters, and for under-regulation by one or more of them.

### Consequences of the Hypothesis

#### Diagnosis

- IBS-B diagnosis is difficult as steatorrhea can be caused by other factors. However the presence of severe symptoms is a very good indicator for IBS-B.
- IBS-C Forms 1 & 2 resemble IBS-A, when the 'IBS Barrier' relaxes quickly & diarrhoea occurs.
- IBS-C Forms 4 & 6 resemble IBS-D, when the mid-abdominal 'IBS Barrier' is not detected and it relaxes soon after breakfast.
- IBS-D Forms 2, 4 & 6 all have very similar symptoms and are difficult to tell apart.

#### Stress and IBS

Environmental and lifestyle factors can cause stress, and when the primary brain controller fails, the resulting IBS symptoms cause more stress. Stress raises the level of activity in the autonomic nervous system by causing the release of adrenal hormones and;

- Any 'IBS Barrier' becomes stronger and constipation, cramping & bloating, are increased in intensity.
- The valve at the end of the small intestine (ICV) becomes easier to open and diarrhoea can occur at any time.

This explains why psychological treatments that reduce stress [5, 6], successfully reduce IBS symptoms.

#### Treatment

Diets free from cereals and whole legumes [4, 5] reduce the symptoms of IBS. If IBS-B or steatorrhea is present then a specially designed diet is necessary to enable fat digestion [5].

If the small intestine becomes acidic, then treatment is more difficult. Proton blockers and a low acid, low fat, low fibre, high carb diet, needs to be eaten [5]. The carbs must be from starchy seeds. Quinoa, buckwheat & amaranth are options.

Relaxation Therapies [5, 6, 7] that teach how to keep the autonomic nervous system at lowest levels are effective at reducing symptoms. They weaken any 'IBS Barrier' & help keep the ICV firmly closed.

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**Table 1:** A summary of the four types and 15 forms of neuro-transmitter deficient IBS, predicted by the hypothesis.

#	Primary controller				IBS Type(s)
	Bile	Duodenum	Jejunum	Ileum	
1	X	O	O	O	B
2	O	X	O	O	C
3	X	X	O	O	C, B
4	O	O	X	O	C
5	X	O	X	O	C, B
6	O	X	X	O	C
7	X	X	X	O	C, B
8	O	O	O	X	D
9	X	O	O	X	D, B
10	O	O	X	X	D
11	X	O	X	X	D, B
12	O	X	X	X	D
13	X	X	X	X	D, B
14	O	X	O	X	A
15	X	X	O	X	A, B

Legend: X = defective and O = functioning